Journal

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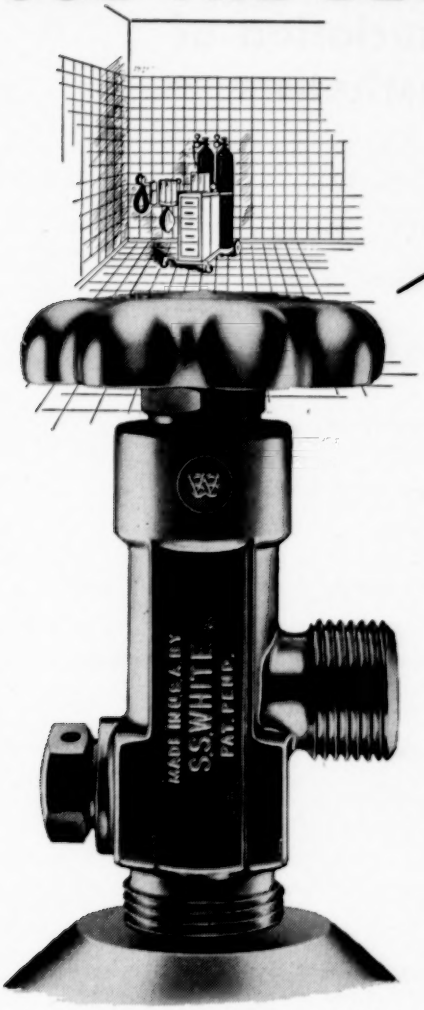

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Twenty-Second Annual Convention American Association of Nurse Anesthetists

September 19-23, 1955

ATLANTIC CITY, N. J.

Hotel Headquarters—Ritz-Carlton Hotel

PROGRAM

Sunday, September 18

8:00 A.M.-5:00 P.M.—**Registration**
A.A.N.A. Registration—3rd Floor Lounge, Ritz-Carlton Hotel

9:30 A.M.-5:30 P.M.—**Registration**
A.H.A. Registration—A.H.A. Headquarters, Traymore Hotel Lobby

9:00 A.M. **Assembly of Directors of Schools of Anesthesia***
Ballroom, 3rd Floor, Ritz-Carlton Hotel
Clarene A. Carmichael, R.N., B.S.
Educational Director, A.A.N.A.
Presiding Officer

Greetings
Minnie V. Haas
President, A.A.N.A.

Introduction to Program
Clarene A. Carmichael, R.N., B.S.

9:15 A.M.-11:00 A.M. **Demonstration on Methods of Teaching**
School Directors Participating

11:00 A.M.-12:00 noon **Comments on Methods of Teaching**
Cameron W. Meredith, Ph.D.
Professor of Education
Northwestern University, Evanston, Ill.

2:00 P.M. **Round Table Discussion**

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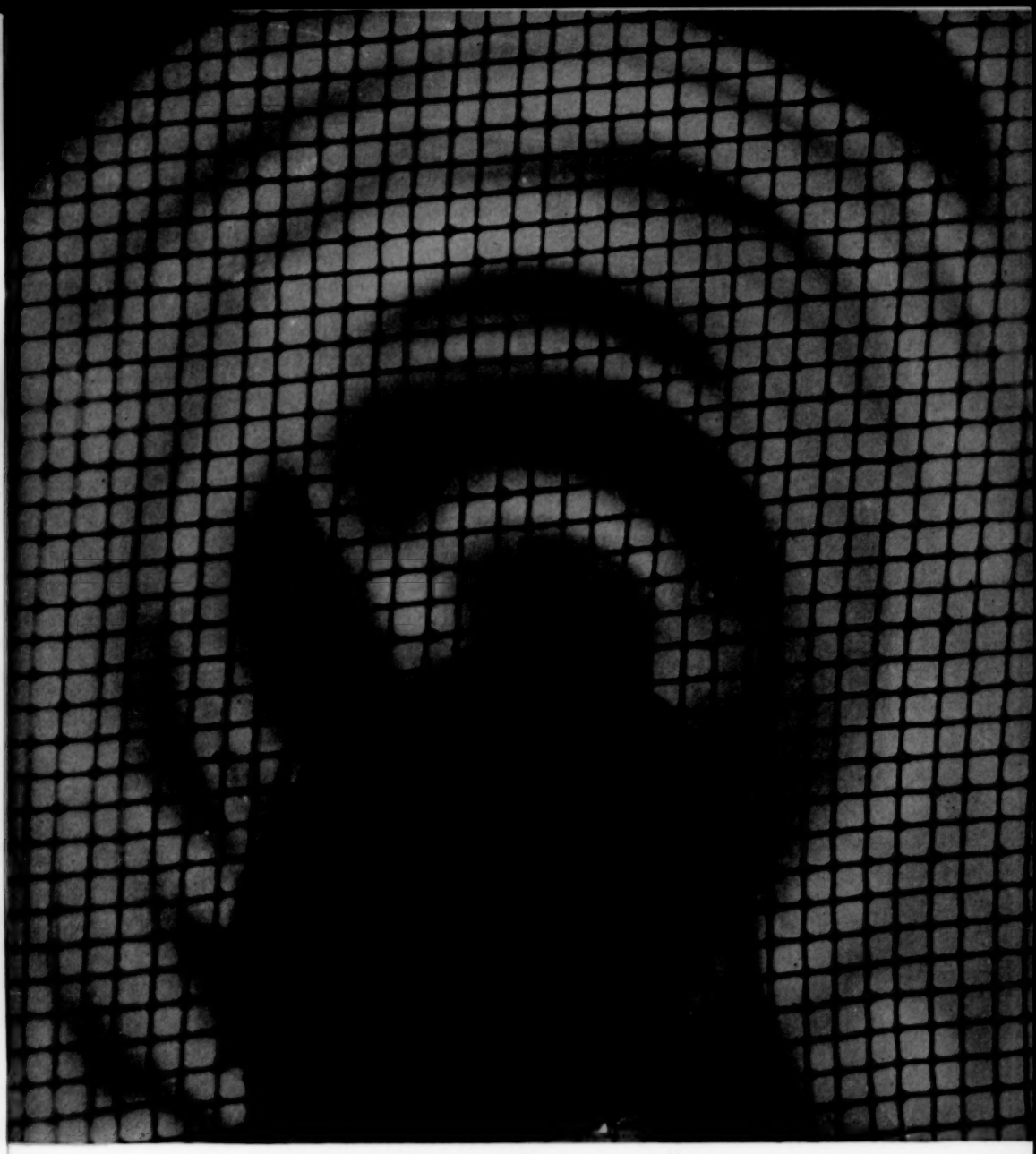
*Although this program is of specific interest to Directors of Schools of Anesthesia, ALL members are invited to attend these sessions.



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Monday, September 19

8:00 A.M. **Registration**
A.H.A. Registration—Convention Hall
A.A.N.A. Registration—A.A.N.A. Exhibit Booth No. 284, Convention Hall

9:00 A.M.-10:30 A.M. **Assembly of Directors of Schools of Anesthesia***
Meeting Room A, Convention Hall
Clarene A. Carmichael, R.N., B.S.
Presiding Officer

Report of Group Leaders from Previous Day's Discussion

10:30 A.M.-12:00 noon **Class Outlines—Distribution and Discussion**
Sample Lectures

2:00 P.M. **General Session**
Meeting Room A, Convention Hall
Minnie V. Haas, R.N.
President, A.A.N.A.
Presiding Officer

Invocation
Alma Prykanoski, R.N.
Trenton, N. J.

Address of Welcome
Minnie V. Haas, R.N.
President, A.A.N.A.

Address of Welcome from A.H.A.
C. J. Foley
Assistant to the Director
American Hospital Association

2:15 P.M.
Doris Nugent, R.N.
President, New York Association of Nurse Anesthetists
Presiding Officer

Deliberate Hypotension
Hrant H. Stone, M.D.
Department of Anesthesiology
Graduate Hospital
Philadelphia, Pa.

Airway Problems in Children
Robert Smith, M.D.
Children's Medical Center
Boston, Mass.

Fatal Circumstances in 384 Anesthesia Deaths
Donald Todd, Lt. (S.G.), U.S.N.
Bethesda Naval Hospital
Bethesda, Md.

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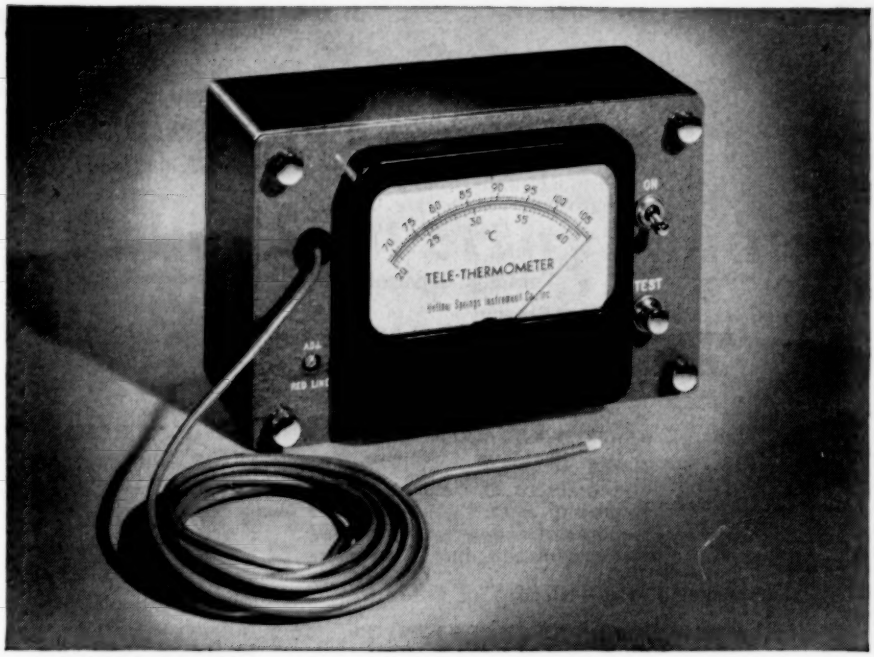

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2. Clark & Trolander, "Thermometer for Measuring Body Temperature in Hypothermia" J.A.M.A., May 15, 1954.

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6:30 P.M. **State Night Dinner**
Crystal Dining Room, Ritz-Carlton Hotel
Pauline Sheppa, R.N.
Chairman, Convention Committee
Presiding Officer

Tuesday, September 20

9:00 A.M. **Business Session**
Meeting Room A, Convention Hall
Minnie V. Haas, R.N.
President, A.A.N.A.
Presiding Officer

Call to Order
Roll Call
Appointment of Tellers
Report of Approval of Minutes Committee
Reports of Officers
Reports of Standing Committee

11:00 A.M.-1:00 P.M.—**Election of Officers**

2:00 P.M. **Business Session**
Meeting Room A, Convention Hall
Minnie V. Haas, R.N.
President, A.A.N.A.
Presiding Officer

Reports of Standing Committee
Reports of Special Committees
Unfinished Business
New Business

Wednesday, September 21

9:00 A.M.-12:00 noon **Council Session***
Meeting Room A, Convention Hall
Florence A. McQuillen, R.N.
Executive Director, A.A.N.A.
Presiding Officer

2:00 P.M. **General Session**
Meeting Hall A, Convention Hall
Anna M. Nagle, R.N.
President, New Jersey Association of
Nurse Anesthetists
Presiding Officer

Anesthesia in Obstetrics—Killer?
James R. Herron, M.D.
Our Lady of Lourdes Hospital
Camden, N. J.

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*Although the Council, as provided by the By-Laws of the A.A.N.A., consists of officers and standing committees of national and state associations, the Council session is open to all members and restricted to members of the A.A.N.A.

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Wednesday, September 21 con't.

Recent Trends in Anesthesia
Robert R. Jones, M.D.
Mayo Clinic
Rochester, Minn.

Newer Trends in Use of Accessory Drugs
Mary A. Costello, R.N.
Cincinnati General Hospital
Cincinnati, Ohio

7:00 P.M. **Banquet**

Crystal Dining Room, Ritz-Carlton Hotel
Minnie V. Haas, R.N.
President, A.A.N.A.
Presiding Officer

Invocation
Reverend Harry Pine
St. Paul's Methodist Church
Atlantic City, N. J.

Address
"The Simplicity to Wonder"
John C. Krantz, Jr.
Professor of Pharmacology
University of Maryland

Thursday, September 22

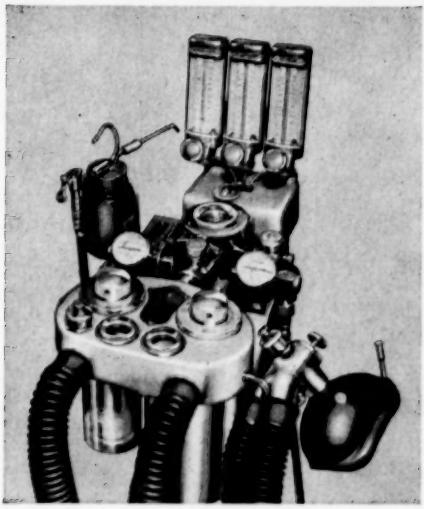
9:00 A.M. **General Session**

Meeting Room A, Convention Hall
Lourene A. George, R.N.
President, Pennsylvania Association of Nurse Anesthetists
Presiding Officer

Problems in Fluid Balance, Pre- and Postoperative Management in Relation to Anesthesia
William T. Fitts, Jr., M.D.
University Hospital
Philadelphia, Pa.

Ether Analgesia
James F. Artusio, Jr. M.D.
New York Hospital
New York, N. Y.

Modern Concepts of Balanced Anesthesia
Francis F. Foldes, M.D.
Chief Anesthesiologist
Mercy Hospital
Pittsburgh, Pa.



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Thursday, September 22 con't.

2:00 P.M. **General Session**
Meeting Room A, Convention Hall
Verna Bean, R.N.
North Wilkesboro, N. C.
Presiding Officer

Sequelae of Endotracheal Intubation
Thomas F. McDermott, M.D.
Professor and Director of Anesthesiology
Georgetown University Medical Center
Washington, D. C.

Neurotics Are People
Hilton S. Read, M.D.
Director, Ventnor Diagnostic Clinic
Atlantic City, N. J.

Treatment of Hypotension When Blood Replacement is Ineffective
Janet McMahon, R.N.
Memorial Hospital
Charleston, W. Va.

4:00 P.M.— **Unfinished Business**

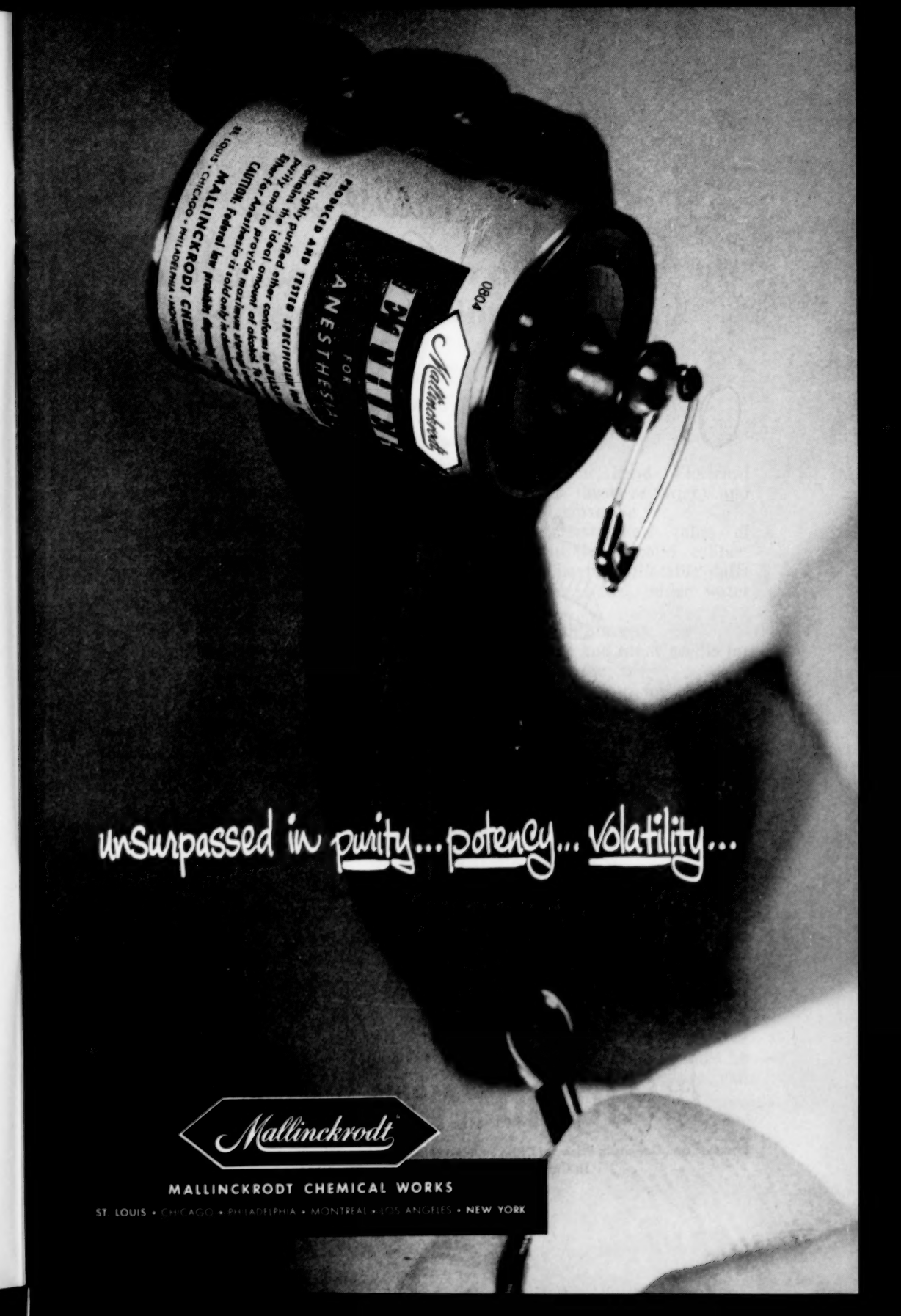
4:15 P.M.— **Adjournment of General Session**

Call to the Convention

As provided for in the Bylaws of this Association, and at the direction of Miss Minnie V. Haas, president, we hereby issue this official call to the members for the annual meeting to be held in Atlantic City, September 19-22, 1955. The annual business session will be held on Tuesday, September 20, in Convention Hall.

Accomplished at the Executive Offices, 116 South Michigan Avenue, Chicago 3, Illinois, this first day of July, 1955.

(Signed) FLORENCE A. MCQUILLEN, R.N.
Executive Director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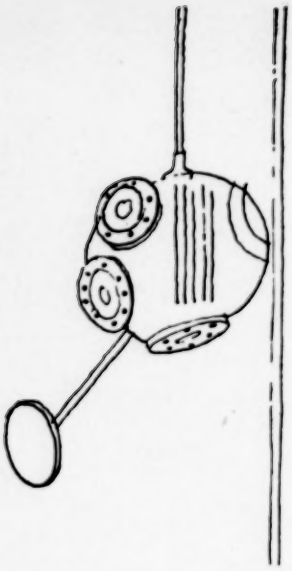

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The Management of Difficult Intratracheal Intubation

Frederick A. Smith, M.D., M.S. *
Cleveland, Ohio

This subject may be a repetition to anesthetists who have had extensive experience with intratracheal intubation, but it is presented for those who haven't had an opportunity to learn the technic, or those who have difficulties on occasion.

The technic properly executed has been almost as important in facilitating the development of surgery and surgical specialties as the introduction of general anesthesia itself. The technic improperly executed can traumatize the airway of patients and can cause serious complications that can discredit the procedure.

It is essential that those performing intratracheal intubation be completely prepared for handling the normal and complicated intubations. The tray (figure 1) would include:

1. A laryngoscope with a blade suitable for the type of patient. The bulb must cast a good light and must not go out when the blade is slapped against the hand. Electrical contacts must be bright and free of corrosion.
2. Two intratracheal tubes of different length and caliber. Those having inflatable cuffs must be tested under water for leaks.
3. A Magill Forceps.
4. A syringe and blunt needle for inflating the cuff.
5. A curved forcep for clamping the air delivery tube to the cuff.
6. Oral and nasal airways.
7. Adequate water soluble lubricant.
8. Suction catheter.
9. 4" x 8" throat packs with fish-line suture tied to each.
10. If a maxillofacial procedure is being done, one must have the nasal intratracheal tubes and proper connectors and adaptors.

Extra intratracheal equipment can be left in the central anesthesia room if someone is free to procure it.

If the anesthetist goes to an area away from surgery, the tray must be complete so that any situation can be handled.

If an anesthetist fails to have the necessary intratracheal equipment

---

*Chief, Anesthesiology Section Veterans Administration Hospital, Cleveland, Ohio.
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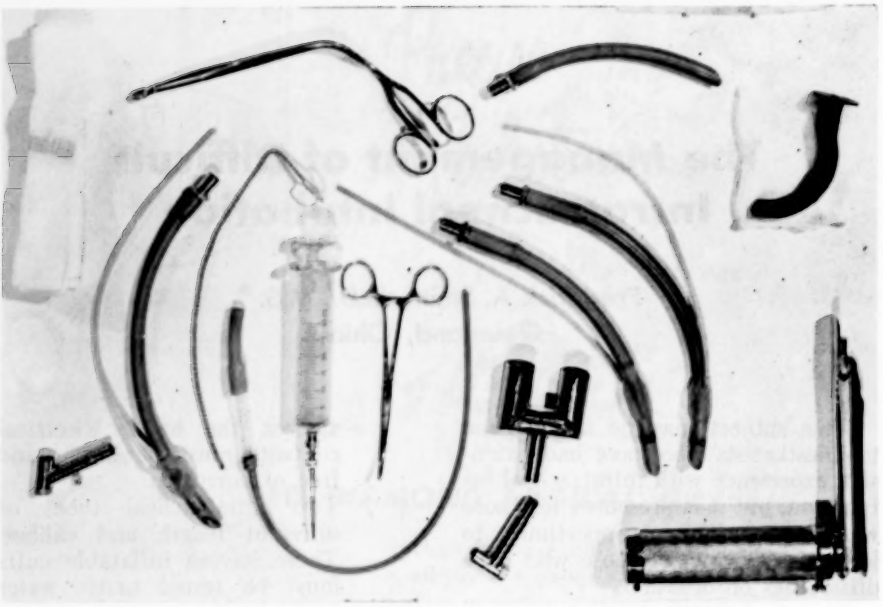


Fig. 1. Equipment for intratracheal intubation

when he sets out to intubate, two serious complications may occur:

1. The protective reflexes may return and the patient will have to be re-anesthetized. The coughing and laryngeal spasm are detrimental to the patient and the delay complicates the operating room schedule.

2. If intubation fails and severe laryngeal spasm develops as with pentothal, or apnea occurs with the use of curare and pentothal technics, a dangerous amount of hypoxia can occur before the airway is re-established. As an example, failure to have the correct size IT tube or a Magill forcep could lead to this complication.

Patients should be deeply anesthetized with ether-oxygen to the third plane, third stage, or, if intravenous anesthesia and curare are used, to an adequate degree of relaxation before intubating. The deeper planes of anesthesia give one time to perform difficult intubations before anesthesia lightens. I personally feel that deep ether-oxygen anesthesia gives the best conditions for intubating. I think that all individuals learning intubation should master the technic under ether before attempting intubation under intravenous anesthesia.

When using a curare preparation to assist intubation, I feel that it is safer to retain some of the patient's ability to breathe, for should a difficult anatomical situation present itself, making intubation impossible, oxygen can still be administered through an oral airway and get to the blood stream. If all voluntary respiration is stopped with curare, and intubation is impossible, tracheotomy may have to be performed to prevent death.

The proper form for intratracheal intubation assists with a normal in-

tubation and allows one to handle the abnormal case with efficiency and without extreme effort.

Fig. 2. (From Lundy, J. S., **Clinical Anesthesia,** W. B. Saunders, 1942.)

Figure 2 illustrates:

1. Opening of mandible with thenar eminence of hand.
2. Eversion of lower lip to prevent blade from catching lower lip against teeth.
3. Lead plate or adhesive to protect incisor teeth and permanent dentures.
4. How the blade is passed in center or to right of center. This position helps to prevent the blade from moving too far laterally and tearing a tonsil pillar.
5. The laryngoscope is held with the left hand. Always pick up the instrument with the left hand and thus save the seconds which some use in passing it from the right to the left hand.

Figure 3 illustrates:

The thumb pushing the tongue to the closed side of the blade. The mandible is pulled onto the blade as the blade is passed downward and forward. It is the opinion of those expert in this technic, that the elevating of the mandible with the right hand is one of the most useful steps in intubation. Note that the mandible is grasped with the thumb inside of the mouth behind the lower teeth. The fingers are on the outside. Care must be taken with patients who have sharp teeth.

Passage of the blade under the larynx and into the esophagus is prevented by observing the base of the tongue first, and the epiglottis second, as the blade is passed down and forward. The blade is passed slowly and deliberately to prevent trauma

Fig. 3. (From Lundy, J. S., **Clinical Anesthesia,** W. B. Saunders, 1942.)

to tonsil pillars, dorsal wall of the pharynx, the epiglottis, ary-epiglottic folds, the posterior commissure or either piriform recess. When intubating, the table should be level. The Trendelenburg position makes intubation difficult because the anesthetist is looking upward and the left arm is higher than the shoulder. The arm gets fatigued because there is less blood supply to the muscles. When intubating, it is of help to rest the left elbow on the table so that the left arm is against the patient's head. This steadies the laryngoscope.

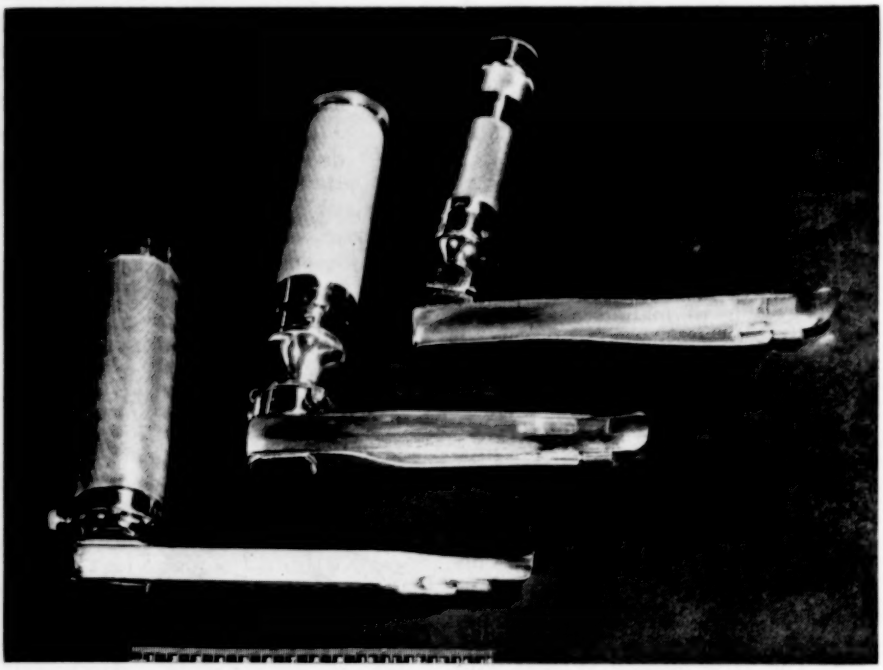


Fig. 4. Laryngoscopes

A variety of laryngoscopes are illustrated in figure 4. They are:

1. The top laryngoscope is Foregger's with a long Guedel blade. This is deep at the handle end and wide open at the top. This blade is difficult to pass through a mouth with poorly relaxed jaw muscles, but is excellent in presence of relaxation because of the wide vision at the larynx.
2. The center laryngoscope, also a Foregger type with a shorter blade, has the same advantages and disadvantages as No. 1. A portion of the blade has been cut away for better vision.
3. The lower instrument, a Flagg type with a 7½″ blade is made by Welch Allyn. This will pass more easily than the other two because it exerts less pressure on the upper teeth. It will

reach into the larynx of a large muscular patient whose jaw is not adequately relaxed. The disadvantage of this blade lies in the fact that the distal end is small and allows very little clearance for the IT tube. (An ideal blade would have the same dimensions at the handle end, but open a little wider at the tip end).

A small Foregger model laryngoscope which is small at the handle end and spacious at the tip end is useful for:

1. Intubating women and teen-age children.
2. Exposing the pharynx for passage of a Levine tube nasally when an IT tube is in place. It occupies less space than a large blade and will not stretch or tear the tonsil pillar.
3. Assisting in intubating patients with arthritis of the temporomandibular joints.
4. Patients with fractures of the mandibles.
5. Patients whose mouths and throats are grossly distorted from recurrent tumors or from radical surgery such as hemimandibulectomy, hemiglossectomy and neck dissection.

I have not had experience with the Macintosh blade, but the long Macintosh blade is advocated by some to be useful in a difficult intubation.

Difficulty in intratracheal intubation may occur in the following situations encountered by anesthetists:

1. Protruding maxillary process with a receding mandible. Many times the maxillary area is narrow also, producing the "squirrel mouth".
2. Protruding maxillary teeth which are long. Long carious teeth are easily broken.
3. Permanent dentures or porcelain caps on maxillary teeth.
4. The large patient with a short thick neck and a small mouth.
5. The patient whose larynx is anteriorly and laterally placed. Many women present this problem.
6. The patient whose epiglottis is of an unusual shape and hard to pick up.
7. Patients with arthritis of the temporomandibular joints, also those with cervical arthritis and resulting deformities.
8. Patients with several upper teeth missing.
9. Patients with fractures of the mandible.
10. Patients who have had radical surgical dissections for carcinoma and have a distorted airway.
11. Patients with cellulitis in areas about the tongue, mandible and neck. Huge carbuncles on the back of the neck cause patient to flex the neck and obstruct the airway when anesthetized.
12. Patients with heavy scarring of face and neck following burns. The scars can prevent normal opening of the mouth.

One of the most difficult problems is the intubation of a patient whose throat is in laryngeal spasm from pentothal. The blood and tissues are cyanotic and if one abrades the structures, dark blood will obstruct the vision. The epiglottis is squeezed over the glottis and the whole larynx squeezed by the pharyngeal constrictors. After lifting the epiglottis, one must twist the tube into the glottis and trachea.

Figure 5 is a photograph of a patient who was difficult to intubate. The side view shows the protruding maxilla and receding mandible.

Figure 6 shows the position of the larynx in relation to the mouth in the same patient.

Figure 7 shows how elevation of the mandible and epiglottis is prevented in this type of patient. The protruding maxillary teeth do not give the blade clearance to pick up the epiglottis. This problem can usually be helped by use of a shallow blade. These will take up less space in the mouth than the deeper blades shown in figure 4.

Fig. 5.

By placing such a shallow blade to the right of center, one can get better exposure of the larynx.

The drawing in figure 8 shows the larynx of a 260 lb. patient who had a small mouth, a long distance to the larynx, and a soft epiglottis. The drawing was made while the patient was in laryngeal spasm. Left drawing shows vocal cords in spasm and epiglottis slipping off the end of the blade. Right drawing shows soft epiglottis collapsing in spasm over glottis. In laryngeal spasm, the constrictors of the pharnyx pull the lateral walls of the pharynx together and squeeze

Fig. 6.

the larynx. The epiglottis gets contracted down over the glottis. If one carefully lifts the epiglottis from the glottis, the patient can inhale and the spasm may break. He can get oxygen until anesthesia is deepened or the IT tube passed. IT tube must be passed with a twisting motion and curve of the tube must be up. Some prefer a straight or slightly curved stilette in the tube. Care must be taken that the stilette does not protrude beyond the tube. These technics are used when one can see only a small portion of the glottis.

Fig. 7.

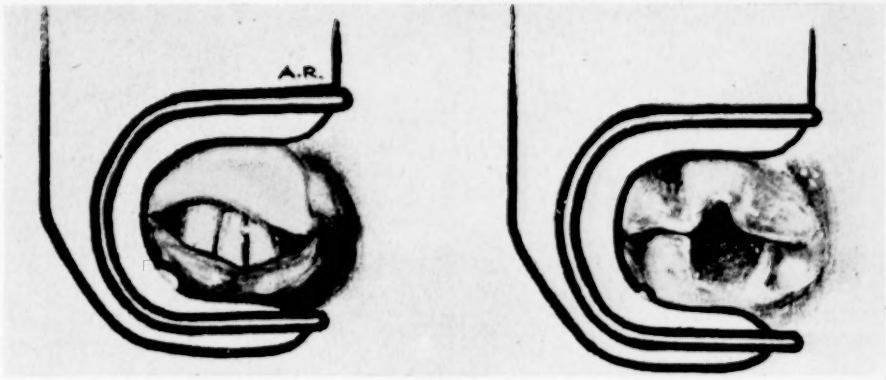
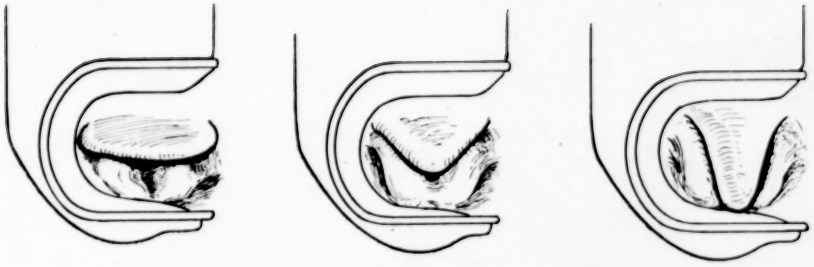


Fig. 8.



(Wide – Stiff – Short) (Short – Thick – Soft) (Long – Narrow – Pointed)

Fig. 9.

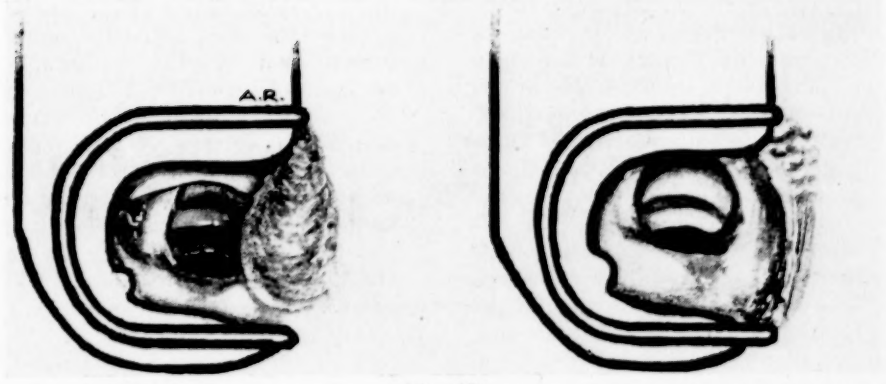


Fig. 10.

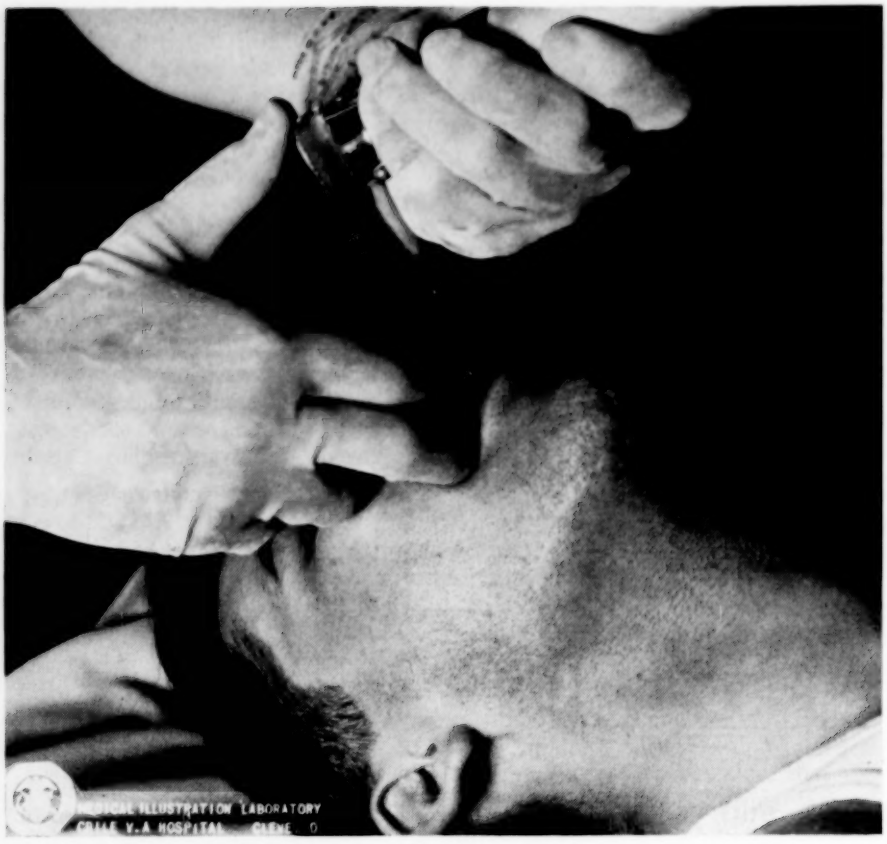


Fig. 11.

In figure 9 drawings show three types of epiglottis which can slip from the end of the laryngoscope blade. The short, thick, soft one can be the most troublesome. It is of help to put the tip of the blade at the aryepiglottic fold and slide to the center. It is easier to handle these under ether anesthesia than under pentothal.

In figure 10 the drawing at the left shows how failure to push tongue to closed side of blade interferes with vision, and the drawing at the right shows how the anterior tonsil pillar can be torn by a large laryngoscope blade. This can happen when the blade is passed too far to the right of the mouth. When an IT tube is in place and a Levine tube must be passed under direct vision, the pillar can be torn because of insufficient space in the mouth for the IT tube, blade and Magill forceps. A torn pillar causes hemorrhage. If a topical anesthetic agent is still present, the denuded area absorbs the drug rapidly. It is of advantage to use the smallest blade possible for this purpose.

Figure 11 shows a conscious patient. In a large muscular patient, where

jaw muscles are poorly relaxed the maneuver illustrated helps to move the blade down to the epiglottis and to elevate it. The maneuver helps after one has elevated the mandible to a maximum with the right hand. Two fingers are placed behind the upper teeth and the thumb pushes on the proximal end of the blade. This maneuver also helps in exposing the glottis of the patient whose larynx is placed anteriorly and laterally.

MISCELLANEOUS

1. **Patients with permanent dentures:** When the laryngoscope must be passed, one can help to prevent breakage of dentures by:

   A deep plane of anesthesia, preferably ether. Jaw must be well relaxed.

   Covering teeth with adhesive or a lead plate molded to shape of teeth.

   Using a shallow blade and placing it laterally to dentures if possible.

   If nasal endotracheal intubation can be done without risk of nasal hemorrhage or kinking of tube, it is wise to do so.

2. **Patients with fractures of the mandible in whom the mouth can be opened only partially:** Surgeons usually like to close the mouth with rubber bands and metal bands after surgery. They prefer to avoid ether because of the postoperative vomiting. A nasal IT tube is needed during the surgery and until the reflexes and consciousness return. In my experience the following technic has been satisfactory:

   a. Anesthetize patient with pentothal and give 100% oxygen until jaws will open one-half inch.

   b. Gently insert a long, shallow blade without disturbing fractured fragments too much.

   c. Using blade as an airway, cocainize base of tongue, pharynx, larynx and as much of trachea as possible. The long DeVilbis atomizer is good for this. Wait one minute for cocaine to act on each area before further inserting blade.

   d. Remove laryngoscope, give a few breaths of 100% O_2, check the blood pressure, and then pass nasal IT tube (well lubricated). Usually it will go into open cocainized larynx, especially when the tube is inserted with the patient's inhalation.

   e. If the nasal intubation fails, pass blade gently and intubate under direct vision. If the patient's jaw gets rigid, use a small amount of a curare which will still allow respiratory movements. Usually this facilitates intubation. If it fails, one still can get O_2 to the patient with an oral airway and try another day with N_2O-O_2-Ether anesthesia or local. Tracheostomy would be used only in the presence of traumatic swelling and obstruction to the airway.

3. **Patients with arthritis of the temporomandibular joints:**

   N_2O-O_2-Ether is preferable and nasal IT tubes go in quite easily. The ether-oxygen gives a large respiratory excursion.

   If the nasal route fails, the ether allows the jaw to be opened enough for the shallow blade to pass. Curare may be of help.

   In patients with deforming arthritis of the neck, the same technic usually is efficient. Some cocainize the nose and throat and pass the IT tube while the patient is conscious, but I have not become adept at this.

4. **Patients who have had radical neck dissections including hemimandibulectomy and hemiglossectomy for carcinoma:** Occasionally these patients come back for more procedures. The airway usually is grossly distorted. Some have recurrent tumor tissue and detritus in the throat. This further obstructs the airway. The tracheostomy is usually healed.

   In the few I have handled I prefer N_2O-O_2-Ether anesthesia with packs to seal mask leaks due to deformity. This technic allows the best respirations without laryngeal spasm.

   The deformed structures allow the use of a shallow blade because it occupies the least space.

   I do not use pentothal and curare intubations for such patients because of the danger of obstruction developing rapidly.

   The technic of passing the nasal tube with cocainization and with the patient conscious, may be preferred by some. However, retained secretions and detritus might be in the piriform recesses and block the blind passage of a tube.

5. **Patients with cellulitis in the mouth, as in peritonsillar abscess or Ludwig's Angina, or cellulitis around the mandible or in the neck:** First it must be determined whether or not there is enough airway obstruction for a tracheostomy.

If tracheostomy is unnecessary, and general anesthesia is used, I have preferred N_2O-O_2-Ether for induction and maintenance. If obstruction occurs early, a nasopharyngeal tube is used. If obstruction becomes severe during induction, a well lubricated nasotracheal tube can be passed. If blind intubation fails, use a long and shallow blade to expose the larynx. Usually ether-oxygen anesthesia causes the least laryngeal spasm without depressing the respiration as do intravenous technics. Postoperatively, the protective reflexes return rapidly. A tracheostomy set always is kept in the room for a case of this type in the event intubation cannot be accomplished.

I wish to repeat two opinions: First, that deep ether-oxygen anesthesia produces the best conditions for intubations and it is best for beginners to learn the technic with ether, and secondly, when using curare, a preparation that allows some respiratory excursion should be used in the event that intubation might be prevented by an unsuspected anatomical deformity.

Hypoventilation in the Anesthetized Patient

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The maintenance of adequate respiratory function or adequate ventilation is of immediate concern to all who practice anesthesia.

Hypoventilation, specifically a decrease in the effective minute volume of respiration, is frequently encountered during the administration of anesthesia. A decrease in minute volume of respiration may occur as a result of decrease in rate, decrease in depth (tidal volume), or as a result of the increase in the size of the dead space. The normal rate of respiration is of the order of 15 to 20 per minute; the average tidal volume is approximately 500 cc. and the average dead space 150 cc. Minute volume of respiration is merely an expression of rate and depth.

$$T.V. \times R = M.V.$$

A patient with an average tidal volume of 500 cc., breathing at a rate of 20 times per minute, will have a minute volume of 10,000 cc. per minute. More important is the effective minute volume of respiration—that amount of air actually reaching the alveoli and participating in the actual gas exchange. Since the air contained in the dead space does not actually participate in this phase of ventilation, effective minute volume may be written:

$$(T.V. - D.S.) \times R = E.M.V.$$

or

$$(500 - 150) \times 20 = 7000 \text{ cc.}$$

Decrease in tidal volume may result in a decrease in effective minute volume since the dead space is constant, even if rate is accelerated. A tidal volume of 200 cc. and a respiratory rate of 40 per minute will result in an effective minute volume of 2000 cc. $(200 - 150 \times 40)$.

The average values for normal adult men are given as approximately 3-4 liters/minute/Sq.M for minute volume, or 2.0-2.5 liters/minute/Sq. M. for alveolar ventilation.

Factors Responsible For Hypoventilation

The following are factors responsible for hypoventilation in the patient:

A. Pulmonary diseases, such as:
   1. Obstructive lesions in the lower or upper respiratory tract.
   2. A decrease in functioning lung tissue caused by atelectasis, tumor, or pneumonia.
   3. A decreased distensibility of

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lung tissue as in fibrosis or congestion.

B. Limitation of the movement of the lungs by pleural effusion or pneumothorax.

C. Limitation of movement of the thorax — arthritis, scleroderma, emphysema, thoracic deformity, or elevation of the diaphragm.

D. Diseases of the respiratory muscles.

E. Interference of neural conduction or transmission.

The role of the above factors as causative agents of hypoventilation is rather obvious. Certainly, when obstruction exists from any cause, air or oxygen may find only limited ingress and alveolar ventilation is impaired. Similarly a lung which lacks normal expansile properties, or a thorax which is not at peak mechanical efficiency, will reduce ventilation. Unfortunately for the anesthetist, many times these are factors over which he has no control but with which he must be prepared to cope during anesthesia and the postoperative period.

The Effect of Position

The time honored positions used for various operative procedures often will result in decreased respiratory efficiency and alveolar ventilation. The assumption of the supine position will reduce both vital capacity and total lung capacity. Many authors have demonstrated reductions in lung volumes in the Trendelenburg (head down) position and in the lateral position with or without the pernicious kidney "rest". Beecher and others have demonstrated poor gas exchange, particularly with regard to decreased CO_2 elimination, during open chest surgery performed in the lateral position. The prone position, often adopted for neuro-surgical operations or proctologic surgery, may severely embarrass respiratory effort and decrease ventilation.

Roentgen examination of the chest with the patient in the lateral position has shown marked congestion of the "down" lung. Such pulmonary congestion might readily interfere with adequate alveolar ventilation, together with disturbances of the ventilation perfusion ratios and intrapulmonary mixing, resulting in ineffective ventilation or gas exchange.

The Effects of Anesthesia

Drugs used for preanesthetic medication.—The drugs most frequently elected to provide preoperative sedation for the surgical patient are the opiates (morphine, meperidine, etc.) and the short-acting barbiturates. These, even when combined with a belladonna alkaloid, frequently result in severe depression of respiration. The actions of morphine as a respiratory depressant are well known, and it has been certainly demonstrated that pentobarbital and its related compounds have a similar effect. As with all narcotics and anesthetic agents, they have the common property of depressing the response of the medullary center to CO_2 the chief stimulus to respiration. Obviously too, the depressant effects are influenced by the dose of the drugs administered, the age and physiologic status of the patient, and the subsequent agents employed for the production of complete anesthesia. Preanesthetic medication must therefore be chosen with care and forethought. Induction of anesthesia with a volatile or gaseous agent may be prolonged and difficult as a result of the hypoventilation brought about by too large a dose of morphine and delayed gas uptake.

Drugs used for anesthesia and analgesia.—The agents used for the production of narcosis are almost without exception depressant to respiration. While it is true that ether in the lighter planes of anesthesia will stimulate breathing as a result of its peripheral action, the drug certainly is depressant in the higher concentrations required for the deeper planes of anesthesia necessary for surgical relaxation. The profound respiratory depression, often to the point of apnea, following induction of anesthesia with cyclopropane and thiopental, are experiences common to all clinical anesthetists. Unfortunately, the gentle to and fro rhythmic excursions of the rebreathing bag of the anesthesia machine are not always reliable guides as to the depth of respiration. While rate may easily be determined, the estimation of depth is not as easy. We have frequently asked resident physicians in anesthesia at all levels of training to estimate the depth and adequacy of the respiratory excursion by watching the rebreathing. Almost always they have over-estimated the quantity of gas moved with each breath. Insertion of a flowmeter into the system has shown them, much to their surprise and chagrin, how inaccurate their estimates were, and proved to them the need to assist or control respirations.

The relaxant drugs and spinal anesthesia.—The introduction of the relaxant drugs ("curare" compounds) has proven in many ways to be a boon to the patient undergoing surgery and has made life perhaps a little more pleasant for the surgeon. Nevertheless, these drugs have created an increased responsibility for the anesthetist, for in producing paralysis of the skeletal muscles of the abdomen, they similarly paralyze the muscles of respiration. They possess no great selectivity of action in blocking one muscle group without affecting another. In our experience, we have found it impossible to secure the desired degree of relaxation of the abdominal muscles without producing at the same time a marked decrease in the respiratory rate and depth. We hold no brief for those who maintain that even with satisfactory muscle relaxation, the patient is breathing adequately. This indicates to us that either the surgeons in other clinics do not require the degree of relaxation demanded in our clinic or that they are not cognizant of existing hypoventilation and its dangers.

Just as the relaxant drugs may produce paralysis of the respiratory muscles, so may the deliberate or untoward administration of high spinal anesthesia result in inhibition of the action of the intercostal and other accessory muscles of respiration. Hypoventilation will ensue and unless recognized and remedied, produce an untoward and sometimes disastrous sequence of events. In our clinic, we have found that approximately one-third of the cardiac arrests that have occurred have resulted under spinal anesthesia. Close examination of the circumstances lead us to believe that in most cases, inadvertent, poorly managed high spinal anesthesia was followed by a period of hypoventilation, decreased oxygenation, and carbon dioxide retention, terminating in cardiac arrest.

The Effects of Hypoventilation

Decreased respiratory exchange produces a twofold effect: one of hypoxia and one of hypercapnia. The latter, retention of carbon dioxide, is certainly less easily recognized and far more insidious.

The dangers of hypoxia have been repeatedly stressed by many authors. The pathologic physiology and clinical signs and symptoms have been repeated in almost every textbook and monograph in anesthesia. The end result of acute and chronic anoxia, ranging from an unexpected emergence delirium to destruction of neuronal tissue and death have been described in detail. Obviously then, it is something to be avoided. To be avoided, it must be recognized but the recognition of clinical hypoxia in the anesthetized, or even sometimes in the conscious individual, may be difficult until real oxygen deprivation exists. This may be particularly true in anesthesia where high oxygen concentrations are supposedly administered. We often sit back in smug security, casting a reassuring eye to see that the oxygen flowmeter is reading properly, only to find that the depressed patient is not breathing deeply enough to clear his dead space and is truly anoxic.

Cyanosis, long regarded as evidence of hypoxia, has been shown by Comroe to be a rather unreliable sign. Physicians of varying degrees of training were unable to correlate skin color and oxygenation in subjects breathing various concentrations of oxygen.

The minute volume of respiration may be effected by breathing oxygen-poor mixtures, but it has been shown that O_2 concentrations must be of the order of 17% or less before significant increases in the minute volume of respiration occur. Similarly the increase in blood pressure, ECG changes, and marked alterations in the pulse rate are frequently indications of severe hypoxia.

When room air is used as the diluent or vehicle for the administration of the anesthetic gases, or if the intraveneous agents such as thiopental are administered alone without supplementary O_2, hypoxia and its symptoms, as the result of hypoventilation, become readily evident. When, however, the patient is breathing O_2, hypoventilation rarely leads to anoxemia but always leads to CO_2 retention.

The problem of carbon dioxide elimination and the effects of hypercapnia have received much attention during the past several years. The oft referred to syndrome of "cyclopropane shock" has been directly related to hypercapnia. The physiologic response of hypotension resulting from CO_2 accumulation, followed by a rather sudden removal of the CO_2, is not alone peculiar to cyclopropane but has been shown to occur as well with ether and thiopental anesthesia.

Intravenous barbiturate anesthesia and those inhalation technics supplemented by a relaxant drug are extremely likely to cause respiratory depression. Gibbon, Beecher, Taylor, and Roos have all shown that severe respiratory acidosis is a common finding in intrathoracic procedures.

The practical aspects of hypercapnia become apparent in the light of recent investigations. Serious cardiac irregularities have occurred following rapid removal of CO_2 from the inspired air.

Hypercapnia is known to potentiate vagal activity. Stimuli such as tracheobronchial aspiration, manipulation above the hilum of the lung, endotracheal intubation or extubation that may otherwise produce no effect, may set up fatal cardiac reflexes. When CO_2 retention is superimposed upon some pre-existing complication of surgery, as infection, toxemia, dehydration, or hemorrhage, tragedies occasionally ensue.

The insidious aspect of hypercapnia lies in our difficulty or failure to recognize its existence. Increases in blood pressure through peripheral constriction may frequently be masked by the anesthesia employed. Changes in ventilation may similarly be masked. Obviously, in the routine practice of anesthesia, one cannot constantly check the pH of arterial blood, nor can one afford, at present at least, to monitor alveolar CO_2 concentrations with expensive analyzers.

How can we as anesthetists solve the problem of hypoventilation? I think at present we recognize the existence of depressed respiration and attempt to correct it by what Beecher has referred to as the "educated hand". In any instances of depressed respiration, either assisted or controlled respiration should be employed. While a great controversy has existed on assisted versus controlled respirations, it has been our practical experience that it is frequently better to institute complete control of respiration than to assist each spontaneous breath. Whenever there exists in the mind of the anesthetist the slightest doubt that ventilation is inadequate, these measures should be employed. Even in the best hands, it is frequently impossible, particularly in certain procedures involving operations within the chest, to insure adequate removal of carbon dioxide. Adequate pressure on the rebreathing bag to maintain good gas exchange may result in forcing the lung into the surgical field. Inadequte pressure resulting in a quiet operative field may lead to marked elevations in the pCO_2 and a severe respiratory acidosis. The fortunate anesthetist is one who can reach a happy medium insofar as ventilation and a good operative field are concerned. We deplore the attitude of what we frequently describe as "no hand anesthesia", in which the anesthetist stands idly by with little or no respect for either the palpating finger on the pulse, or for the hand on the rebreathing bag to assist respirations in a depressed patient. Until we have available adequate mechnical means for maintenance of respiration in the unconscious patient, or until we have available anesthetic agents which will not directly or reflexly depress respiration, the maintenance of adequate ventilation is a function of minute-to-minute control and the careful and well regulated use of the "educated hand".

Coagulation Failure and a Hemorrhagic State Occurring During an Operation

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The anesthetist has to share many experiences with a surgeon during the course of an operation. Most of these experiences are pleasant and gratifying, but occasionally one may be frightening. A potentially dreadful experience can be the development of a hemorrhagic disorder. Fortunately, this complication occurs infrequently but when it does develop, it is usually unpredicted and unexpected. It is manifested by the onset of uncontrollable oozing from the surfaces of the operative wound during the operation or shortly following the conclusion of the procedure. The outcome may be fatal as a result of hypotension secondary to blood loss unless the hemorrhage can be controlled locally or unless the patient can be maintained until treatment and readjustment of the disordered physiology can arrest the bleeding. There is no doubt that this hemorrhagic state signifies a failure in hemostasis, but in only about 50% of the cases can the cause of the failure be identified. Our inability to discover the source of the defect in the remaining 50% of the cases is related to the complexities of the blood clotting process and to the lack of dependable methods for measuring the effect of the individual factors involved in hemostasis.

Why blood remains fluid within the vascular system and yet normally undergoes clot formation when this system is interrupted has been a challenging question for years. In recent years it has become possible to sketch a plausible sequence of changes involved in hemostasis, and though a number of mysteries remain. this increased understanding of the hemostatic process has permitted the identification of the defects producing a variety of hemorrhagic diseases. Since uncontrollable hemorrhage is a possible hazard in any operation, the surgeon always attempts to discover pre-operatively whether a bleeding tendency exists. Fortunately, the history presented by the patient, plus a few laboratory tests, usually suffice to give gross evidence of a normal clotting mechanism. Since most hemorrhagic disorders give rise to bleeding episodes without relation to an operation, it is usually possible to investigate the nature of the disease before an operation and thus the surgeon is able to modify his recommendations to the patient. Though the majority of hemorrhagic diseases can be recognized before an operation, there are a few bleeding disorders which develop acutely during an operation or in the immediate

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post-operative period and which are the result of an acquired defect in the clotting process produced by the stresses of the operation or the disease necessitating the operation. It is this latter group of hemorrhagic states that will be the subject of the present comments.

Before undertaking the discussion of the possible causes of such defective hemostasis, it is pertinent to review the factors involved in the natural control of hemorrhage. (Table I)

Table I
FACTORS INVOLVED IN HEMOSTASIS

I. Extravascular
   A. Vessel Location v.s. Tissue Support
   B. Extravascular Tissue Tension
   C. Tissue Thromboplastic Activity
II. Vascular
   A. Structure of Vessel Wall
   B. Physiologic Attributes
      1. Contractility
      2. Retractility
      3. Fragility
      4. Permeability
III. Intravascular
   A. Hemostatic Platelet Plug
   B. The Coagulation Process
   C. Inhibitors of the Coagulation Process

After an injury is inflicted, a sequence of changes occurs as a result of the influences of extravascular factors derived from the damaged tissue, of vascular factors inherent in the response of the severed blood vessel wall, and of intravascular factors related to the coagulation mechanism of the blood. This is a dynamic chain of events in which normally the individual elements cannot be isolated so that in any one area of injury various steps in the hemostatic process exist simultaneously.

Bearing in mind the important concept of physiological motion with regard to the natural control of blood loss, a resume of the extravascular factors provides a logical starting point. A superficial vessel overlying a rigid supporting structure such as bone is particularly vulnerable to injury, whereas a vessel protected by relatively rigid structures is less likely to be injured. Vessels situated beneath the epithelium of body spaces, such as the nose and bowel, can bleed into these spaces with little resistance. Bleeding into fat or loose areolar tissue may be unimpeded for some time in contrast to bleeding into the fascial sheath of a muscle where the fascia effectively resists distention and tends to limit the extent of the hemorrhage. In addition to the anatomical factors involved, damaged tissues provide a chemical source for activation of the clotting mechanism in the blood. Though we are speaking of hemostasis in a positive direction, it should be pointed out that these same damaged tissues can be a source of inhibitors or destroyers of clotting.

The injured blood vessels play an important role in hemostasis. The anatomical structure of the vascular wall influences the type of injury, the nature of the bleeding, and the response of the vessel to the injury. Thus thick-walled muscular arteries are better protected from injury than the thin-walled friable veins. However, venous bleeding is more likely to be controlled spontaneously or by pressure because the column of blood flowing through veins is under low pressure. The endothelial lining of the blood vessel favors the fluid state of the blood, and when it is disrupted, the cells tend to swell and changes occur which permit the blood platelets to clump and become attached to the site of injury. The functional capacity of vessel walls affects hemostasis. When a vessel whose wall contains muscle is severed, there is a transient contraction of the lumen and the elastic fibers in the wall shorten, retracting the free end of

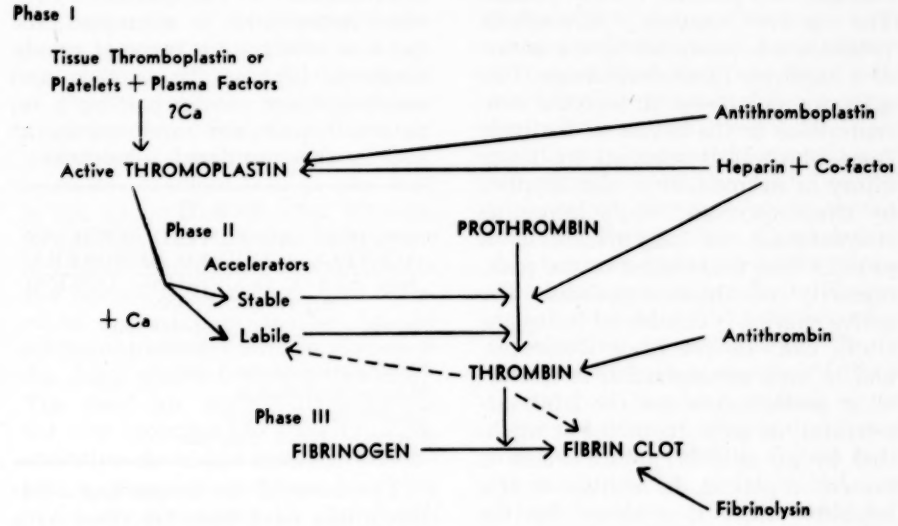
the vessel. These responses favor the action of the extravascular and intravascular factors in producing a hemostatic clot. Fragility is a property related to the structure of the blood vessel wall which expresses the resistance of a given segment of vessel to disruption from increases in intravascular pressure. Thus the smaller thin-walled arterioles and venules are more likely to rupture when confronted with local increases in internal pressure than are the larger muscular well-supported vessels such as the main systemic arteries which are designed to withstand the full force of the heart's action. Permeability is the final independent property of a blood vessel wall. This term describes the function which permits the exchange of substances between the blood and the fluid medium outside of the blood vessels and body cells—the interstitial fluid. The term is employed in reference to the capillary bed where the endothelial cells and intracellular cement control the exchange. The capillary wall is very sensitive to changes in physical and chemical environment to which it responds by changes in permeability. Although the site of bleeding in a hemorrhagic diathesis has not been definitely localized, the capillary bed or immediately adjacent arterioles are usually indicted because marked increase in permeability and minute ruptures of the vessel wall allow the escape of blood.

The intravascular factors complete the process of hemostasis. The blood platelets appear to be essential to the integrity of the capillary wall. They have unique property of clumping or agglutinating at the site of an opening, preventing loss of blood; and when a blood vessel is injured, the platelets mass to form a hemostatic plug. As some of the platelets are disrupted in this process, a substance is released which produces a chemical vasoconstriction perpetuating the original contraction of the vascular wall which was produced by the injury, and another substance is released which combines with certain plasma factors to initiate the clotting process. A final function of platelets concerns their part in the retraction and anchorage of the fibrin clot which is laid down as the result of blood coagulation. The dynamic features typifying the whole hemostatic process are emphasized in the scheme of coagulation. (Figure I)

As it is outlined in skeletal form, coagulation occurs in phases of changes which are antagonized by inhibitors so that the balance between accelerating factors and inhibitory substances determines whether there will be a clot or perhaps uncontrollable oozing from interrupted vessels. Initially, active thromboplastin evolves from substances introduced by the tissues or platelets plus factors normally present in the plasma. The thromboplastin, with the help of calcium ions, activates the two accelerator systems which convert prothrombin to thrombin. This enzyme then produces a fibrin clot from fibrinogen. The plasma normally contains the plasma factors of the first phase, among which is anti-hemophilic globulin. Also the precursors of the labile and stable accelerators of the second phase, prothrombin, and fibrinogen, are present in the normal plasma. In addition, antithromboplastin, antithrombins including heparin, and fibrinolysin (plasmin) exist in the plasma in active or inactive precursor states ready to limit the extent of blood clotting within the vascular tree.

Figure I

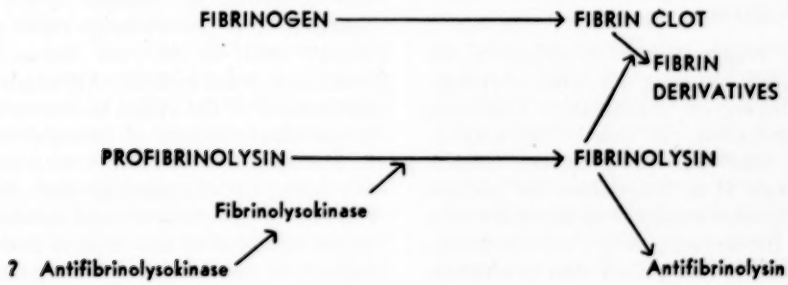
COAGULATION SCHEME
DYNAMIC SYSTEM



The fibrinolysin system is the important mechanism available to the body for destroying clots. (Figure II) It is responsible for the liquefaction of extra-vascular clots, and, though its function in the plasma is not clearly defined under normal conditions, it logically plays a role in limiting the extension of intravascular thrombi which form to plug the small leaks which develop continually and spontaneously. There is good evidence that the various coagulation factors are being used up and replenished continually, supporting the idea that thrombi are being formed to protect the intregrity of the vascular tree. The fibrinolysin-antilysin system, as

Figure II

THE FIBRINOLYSIN - ANTIFIBRINOLYSIN ENZYME SYSTEM



it is currently understood, rivals the complexity of the coagulation scheme. A precursor, profibrinolysin (plasminogen) is present in the plasma. The active enzyme, fibrinolysin (plasmin), is produced by the action of a catalyst, fibrinolysokinase. This activator is present in varying concentrations in the tissues of the body from which it is released by tissue injury or necrosis. It is also supplied by streptococci and staphylococci as streptokinase or staphylokinase—a product that contributes to the pathogenicity of these organisms. The active enzyme is capable of lysing the fibrin clot, its precursor fibrinogen, and in high concentration it can destroy prothrombin and the labile accelerator, as well. Its action is inhibited by an antifibrinolysin which is present in plasma. In addition to this inhibitor, there is evidence for the existence of an inhibitor of the fibrinolysokinase in tissue.

This brief review of the factors involved merely stresses the point that the spontaneous arrest of bleeding results from the synchronous action of all of the factors. Furthermore, in support of clinical observations, it is evident that severe defects in hemostasis can exist with no recognizable abnormality in blood coagulation while, on the other hand, serious impairment in blood coagulation may not be manifest in a disorder of hemostasis in the absence of unusual stress.

To return to the problem of an acquired bleeding tendency developing during or immediately following an operation, it should be emphasized that this complication usually develops at a time when the patient shows other evidence of not tolerating the procedure very satisfactorily. When it is recognized that uncontrollable oozing is present, it is difficult to decide whether the hemorrhagic disorder is the cause or effect of the deterioration of the patient. Then when resuscitation is attempted, the cause or effect issue becomes purely academic because the surgeon and anesthetist are usually battling a resistant hypotension and anoxia as well as the disordered hemostasis.

Table II

FACTORS ASSOCIATED WITH AN ACUTELY ACQUIRED HEMORRHAGIC STATE OCCURRING DURING AN OPERATION

Anoxia - Hypoxia (Hypotension)
Depression of Liver Function
Manipulation of the Lung
Massive Transfusion
Transfusion Reaction
Cancer - Tissue Necrosis

The cases of the hemorrhagic diathesis that have been described by a number of observers have been associated with a series of circumstances that are not uncommon problems to the anesthetist or surgeon. (Table II) It will be evident that a number of the factors may be operating at the same time; in fact, it is frequently difficult to separate the physiologic consequences of one factor from another. Thus a period of hypoxia or anoxia is almost invariably associated with the onset of uncontrollable bleeding. Hypotension accompanies or contributes to the oxygen deficit, and the net result of this situation is cellular injury or death of the particularly vulnerable tissues such as nervous tissue, the liver, and heart muscle. Although the mechanism of the effect on hemostasis is not clear, failure of transportation of the coagulation factors as a result of impaired circulation, the introduction of accelerators and inhibitors as an outcome of cell injury, and the failure of production of coagulation

factors because of damage to the source of supply may all lead to hemostatic failure. Preoperative liver dysfunction may be intensified by the depression of liver activity produced by general anesthesia and consequently interfere with the supply of the coagulation factors. Transthoracic procedures have been responsible for a number of hemorrhagic states. Manipulation of the lung is apt to produce or abet hypoxia. In addition, the fact that lung tissue is a good source of thromboplastin and fibrinolysokinase has been offered as a partial explanation for the relationship between manipulation of the lung and a bleeding tendency. The need for massive transfusions not only indicates the severity of the operative stress but may also contribute to coagulation defects by virtue of the loss of the unstable coagulation factors attending the storage of blood. Storage of blood leads to prompt disappearance of the platelets, progressive loss of prothrombin and consequently thrombin activity, and a decrease in the accelerators of the first and second phases of the coagulation process. If a few clots form because of failure of complete admixture of the anticoagulant solution during the process of drawing the blood, there is further loss of the factors just mentioned plus consumption of some of the available fibrinogen. The inactivation of calcium ions by the citrate of the storage solution has been indicted as a source of difficulty, but this chemical reaction has not been proven to be a limiting factor in the behavior of the coagulation process in massive transfusions. A transfusion reaction may contribute to a hemorrhagic state by inducing antibody production after sensitization has been developed from previous transfusions, by contributing to the release of circulating anticoagulants which remain ill-defined, or by affecting the integrity of the small blood vessels. The exact mechanisms of the effects of transfusion reactions are not well understood but it cannot be denied that they can contribute to a bleeding tendency. Finally, tissue death or abnormal neoplastic tissue growth may be related to an acquired hemorrhagic condition that becomes apparent at the time of or shortly following an operation. This relationship has been explained by the release of substances that accelerate fibrinogen conversion—such as thromboplastin—leading to a deficiency in fibrinogen through formation of fibrin thrombi; it has been related to the abnormal release of fibrinolysokinase which activates the lytic system leading to destruction of fribin clots; or it may be connected with other factors which have not been identified as yet. Admittedly these explanations are in part conjectural. The influence of neoplastic, inflammatory, or necrotic changes in tissues on the coagulation mechanism becomes quite confusing when it is pointed out that hypercoagulability of the blood and venous thrombosis not infrequently accompany the same tissue changes.

It is evident that the aforementioned circumstances occurring with a failure in hemostasis are not mutually exclusive so that any time this complication develops, one or more of these conditions may be contributing to the uncontrollable bleeding. In spite of this fact, it is possible to outline the basic defects in hemostasis that potentially could give rise to a hemorrhagic state. (Table III) Deficient thromboplastin activity is produced by unopposed circulating anticoagulants which may result from anaphylactic shock or a transfusion

reaction. Interference with thrombin formation is related to a reduction in available prothrombin or the prothrombin-thrombin conversion accelerators which may result from preexisting liver disease or Vitamin K_1 deficiency or may be produced by massive transfusion of stored whole blood. The fibrinogen available for clot formation may be reduced by failure of formation, as in liver disease and massive transfusions, by destruction of the fibrin clot or fibrinogen from excessive fibrinolytic activity, or by excessive thromboplastic defibrination which produces widespread fibrin deposition without relation to orderly hemostasis. And, finally, defective coagulation may be combined with vascular abnormalities induced by allergic phenomena which are poorly understood.

Table III

DEFECTS RELATED TO AN ACUTELY ACQUIRED HEMORRHAGIC STATE

I. Defective Coagulation
  A. First Phase - Deficient Thromboplastin Formation
    1. Circulating Anticoagulants
      a. Anaphylactic Shock
      b. Transfusion Reaction
  B. Second Phase - Deficient Thrombin Formation
    1. Hypoprothrombinemia
    2. Deficiency of Stable or Labile Accelerators
      a. Liver Disease
      b. Vitamin K_1 Deficiency Formation or Absorption
      c. Massive Transfusion
  C. Deficient Fibrin Clot
    1. Hypofibrinogenemia
      a. Liver Disease
      b. Massive Transfusion
      c. Excessive Fibrinolytic Activity
      d. Excessive Thromboplastic Activity —Defibrination
II. Defective Coagulation plus Vascular Abnormalities
  A. Vascular Purpura
  B. Anaphylactoid Purpura
    1. Transfusion Reactions
    2. ? Antiplatelet Factor
    3. ? Thrombasthenia

The treatment of a hemorrhagic state acquired during the operative period includes supportive measures which are universally used in treating hypoxia or blood loss from any cause and measures designed to correct the hemostatic defect. The latter measures presuppose identification of the defect which, as noted earlier, is not possible in approximately 50% of the cases. Further, in a number of instances in which the defect is finally demonstrated by means of various laboratory tests, the information is not useful practically in managing the individual patient because the time required to accomplish identification is too long or no specific means is available for counteracting the cause of the hemostatic failure. Thus, it is necessary to have a plan of treatment that can be initiated as soon as the hemorrhagic state appears.

The primary aim is to sustain the patient until the process can be halted by the body and/or some supplementary specific measure. The importance of maintaining oxygen supply and supporting oxygen and carbon dioxide exchange is unquestioned. The considerations involved are, of course, assuring effective ventilatory exchange, protection of functioning pulmonary tissue, maintenance of cardiac action, support of the vascular tone, and provision of erythrocytes for oxygen and carbon dioxide transport. Some of the corrective measures are well established as beneficial in the treatment of a hemorrhagic state while others offer uncertain help. Removal of a neoplasm, inflammatory tissue, or dead tissue can eliminate one factor contributing to the bleeding. Whole blood is employed universally to maintain an adequate blood volume in the face of

blood loss, and fresh whole blood is a dependable source of the coagulation factors necessary for hemostasis. Similarly, fresh plasma supplies functional coagulation factors, but it is not as desirable as whole blood in an acute bleeding episode because of the absence of red blood cells. Serum may supply some antilysin in addition to its temporary colloid osmotic effect, but the actual amount of antilytic activity is quite variable and not dependable. Fibrinogen is valuable in those instances in which a fibrinogen deficit exists as a result of its destruction or utilization. Vitamin K_1 can be given intravenously as an emulsion without fear of adverse effects when used in the usual 50 milligram dose. Protamine sulfate and toluidine blue are potent antagonists of heparin and heparin-like substances, but in the hemorrhagic state the action of these substances is difficult to assess. If the anticoagulant effect in the abnormal blood can be reversed in a sample of the blood by either of these substances, it would be worthwhile to administer one of them. There is little doubt of the importance of vitamin C in the function of the intercellular cementing substances which is vital to the integrity of the capillary and arteriole walls, and, consequently, administration of vitamin C may be helpful. The position of adrenochrome is not clear, though evidence indicates that it may play a role in the vascular factors of hemostasis. Furthermore, it is relatively non-toxic so that its empiric use is not hazardous when a vascular defect is believed to exist. Corticotropin, hydrocortisone, and cortisone have been effective in controlling some hemorrhagic states associated with antibody reaction and "overactivity" of the spleen. It appears that the steroids can affect intravascular and vascular factors in hemostasis, but the exact position of these substances is not clear. They can be used safely in acutely acquired adrenal insufficiency and may be helpful in the hemorrhagic state associated with an allergic reaction.

This review of the hemostatic process and of the possible causes of an acute hemorrhagic state arising during an operation was designed to emphasize the aspects of the problem that are understood and to point out the unknown facets that still exist. A program of treatment is offered for your consideration with the hope that it can be revised in the future to include more specific corrective measures. Though a fatal bleeding state is infrequent, it behooves us all to be aware of the condition so that early recognition and initiation of treatment will give the affected patient the best possible chance to recover.

Pulmonary Function

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Pulmonary function as a unit includes adequate oxygen supply to the tissues as well as the elimination of carbon dioxide and the maintenance of a pH of 7.40 in the arterial blood. In discussing pulmonary physiology and some of the tests used to study the pulmonary function, I shall use the nomenclature agreed upon in 1950 and published in the Federation Proceedings of that same year (9: 602-605, 1950).

I would furthermore like to point out that all tests discussed show us some aspect of the patient's pulmonary function—they never explain why this function is disturbed in a certain way. In other words, we still have to make a clinical diagnosis before we can evaluate the deviation from the physiological norm.

Pulmonary function will be easier to study and to understand if we break it down into several separate phases, always realizing that these present but different facets of one unit.

We distinguish between ventilation, diffusion or respiratory gas exchange and pulmonary capillary blood flow, and we shall discuss them in this order.

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Ventilation

Ventilation is the movement of gas mixtures, like atmospheric air or anesthetic mixtures, into the lungs and out of them and the distribution of these mixtures within the alveolar space. It represents that part of pulmonary function which is most familiar to us, so familiar indeed, that we are likely to overlook the importance of the other two functions. Effective and efficient ventilation requires first of all patent, unobstructed air passages, an intact, freely expandable chest wall, a stable mediastinum, a properly functioning neuromuscular apparatus and normal elasticity of the lung tissue. Any deviation from normal in any of these structures tends to interfere with the mechanics of ventilation, leading to inadequate oxygenation, carbon dioxide retention and a pH shift towards acidosis.

We all know the deleterious effects of an obstructed airway, of a wide open chest and a swinging mediastinum. We might not see a patient with poliomyelitis every day, but the effects of the neuromuscular paralysis of a high spinal anesthesia impair ventilation just as thoroughly as does muscle paralysis from curare-like drugs or excessive depth of anesthesia and premedication. These are all conditions we are trained to detect. We should be capable of remedying them. Other conditions, like emphysema or

pulmonary fibrosis, are less dramatic, but they interfere with ventilation and reduce the reserve not any less than the other disturbances just mentioned.

It would be particularly desirable for anesthetists to know before surgery the status of their patient's pulmonary function and the degree of pulmonary insufficiency present. Dyspnea is the leading sign of ventilatory insufficiency, but it is a subjective sign, present only with pronounced failure and masked by heavy sedation or general anesthesia. There are, however, tests that will enable us to analyze the ventilation and detect mild degrees of insufficiency before failure becomes clinically manifest under the stresses of surgery and anesthesia.

We know today that the measurement of the lung volumes that has been praticed for a long time is in itself not adequate to evaluate ventilation. Nevertheless, these measurements provide us with a certain baseline for comparison and evaluation of other tests. We distinguish between lung volumes that do not overlap and lung capacities that include two or more of the volumes.

Tidal Volume—the amount of gas mixture inspired or expired during each respiratory cycle. (Average adult: 500 ml.)

Inspiratory Reserve Volume—the maximum amount of gas that can be inspired from the resting end-inspiratory position. (3100 ml.)

Expiratory Reserve Volume (formerly supplemental air)—the maximum amount that can be expired from the resting end-expiratory level. (1200 ml.)

Residual Volume (formerly residual air)—the amount of gas remaining in the lung after maximum expiration. It cannot be expelled unless the chest wall is open or the intrapleural pressure is increased as in pneumothorax. (1200 ml.)

Total Lung Capacity—the amount of gas in the lung at the end of a maximum inspiration. (6000 ml.)

Vital Capacity—the maximal amount of air that can be expelled forcefully after a maximum inspiration. (4800 ml.)

Inspiratory Capacity (formerly complemental air)—the maximum that can be inspired from resting expiratory level. (3600 ml.)

Functional Residual Capacity—the amount of gas remaining in the lung at the end of the resting expiration. (2400 ml.)

The residual volume, the functional residual capacity and the total lung capacity cannot be measured directly, but have to be calculated by measuring the concentration of nitrogen in the expired gas mixture after breathing 100% oxygen for 7 minutes. The other volumes and capacities can be measured directly. Deviations from the average might be significant; normal values, however, can be present with marked ventilatory insufficiency. Patients with asthma or pulmonary emphysema, for instance, might have normal tidal volumes and vital capacities with only an increased functional residual capacity and a reduced inspiratory reserve volume and be completely disabled. The reason for this: none of the tests described so far pay attention to the time element, the speed and the ease of moving gas volumes

into the lungs or out of them. A patient with a closed pneumothorax might have a marked reduction of his vital capacity and total lung capacity, but with normal tidal volume he might be capable of moderate efforts without dyspnea.

Fortunately, there are simple volume-per-time measurements available that permit us to obtain a rather rapid, reliable estimate of our patients' ventilatory reserve. One is the "timed vital capacity"—it gives us the fractions of the vital capacity, expelled at the end of 1, 2 and 3 seconds. Normally 83% should be expelled at the end of 1 second, 97% after 3 seconds. Obstructive lesions, like asthma, greatly reduce the fractions expelled during the first second, because the free gas flow is greatly impaired in this condition. In evaluating patients for thoracic surgery, two other tests proved particularly valuable to me. They are the walking ventilation (WV) and the maximum breathing capacity (MBC), one measuring the amount of air exhaled during moderate exercise (walking for three minutes at a speed of 180 feet per minute), the other measuring the air volume expelled during vigorous and forceful inhalation and exhalation (for 15 or 30 seconds), and both calculated as output per minute. The drawback of these tests, especially MBC, is the necessary cooperation of the patient. We may, however, safely assume that a surgical patient will more readily cooperate than one with compensation claims.

Patients with marked ventilatory insufficiency might, of course, not be capable of undergoing these tests without severe dyspnea. However, you cannot subject patients unable to tolerate the tests to surgery or anesthesia as they have no pulmonary reserve of any significance. The walking ventilation gives a fairly constant value, around 10 to 20 liters per minute, and is an excellent base line to evaluate ventilatory or overall pulmonary efficiency and economy. It does not change in the presence of pulmonary pathology unless there are extensive changes and destruction of the pulmonary tissue. The maximum breathing capacity depends on sex, age, and body surface and indicates the upper limits of the pulmonary reserve. It has been shown that the index of walking ventilation over maximum breathing capacity gives us a quick estimate of the ventilatory reserve. Patients whose index WV/-MBC approaches or exceeds 0.5 are severely dyspneic and cannot tolerate extensive surgery. The normal index is about 0.2, and patients whose index does not exceed 0.3 are usually good surgical risks. About 80% of our patients can be rapidly and adequately evaluated with walking ventilation and maximum breathing capacity. It is the remaining 20%, the borderline cases, that need much more thorough and elaborate studies, not only of the mechanics of ventilation, but also of the oxygen uptake, the functional residual capacity, and the oxygen saturation as well as the pH of the arterial blood.

In patients scheduled for pulmonary resection, we are, in addition, concerned with the function of each individual lung. Divided endobronchial catheters, like Carlens' catheter, which you may have used for anesthesia, were originally designed for such simultaneous bronchospirometric function studies of the separate lungs. One part is introduced into the left mainstem bronchus and sealed off with an inflated cuff; the other half terminates in the trachea with its separate endotracheal cuff, and collects the air from the trachea and

the right lung alone. Bronchospirometry provides valuable information, but it involves obstructive breathing through narrow tubes. We found the Carlens tube life-saving in two cases of massive pulmonary hemorrhage, but we feel that other endobronchial tubes, like the Bonica tube, produce less obstruction (if any) and resistance. The great advantage of the Carlens tube lies in the facility of more or less blind intubation, which is of importance in massive pulmonary hemorrhage. We cannot recommend its routine use for anesthesia until a new design with a larger lumen reduces resistance to airflow. The resistance is large enough to limit evaluation of bronchospirometric data to a comparison between right and left lung. Normally about 55% of the ventilation and the oxygen uptake are carried by the right lung, 45% by the left. When, for example, bronchospirometry and fluoroscopy show that one side carries 80% of the function, then we can safely assume that resective surgery on the other side not only will not impair, but might even improve overall function. On the other hand, surgery, no matter how urgent, is impossible if the side carrying 80% of the function would even temporarily be put out of commission by a thoracotomy.

If a hospital does not have an elaborate physiology laboratory available, some rough estimate of the ventilation can be obtained by having the patient observed before the fluoroscope. The degree of freedom of movement of the ribs and the diaphragm, the speed of aeration during inspiration, and airtrapping during expiration can easily be estimated before the fluoroscope. Usually a chest physician or a thoracic surgeon will be most proficient at evaluating these observations. We have occasionally used the clinical trial of anesthesia to estimate the effect of a proposed pneumonectomy. We introduce a Bonica tube into the mainstem bronchus of the lung that would have to carry pulmonary function during and after surgery. If a rapid deterioration of the patient is observed after exclusion of the "operative" lung it would be wise to advise against surgery. These tests are not as elegant and convincing as bronchospirometry or arterial oxygen determination, but they might prove helpful where no other way of evaluation is available.

In discussing ventilation we should at least briefly mention the anatomical dead space. With a tidal volume of 500 ml. and 150 ml. dead space, we have an effective alveolar ventilation of 350 ml., which, at a rate of 16 breaths per minute, would make for a minute volume of 8000 ml. and a minute alveolar volume of 5600 ml. A tidal volume of 250 ml. with a rate of 32, as might be present in a depressed patient, will produce the same minute volume, but the effective alveolar volume would be reduced to 3200 ml. This disproportion between dead space and tidal volume is one of the reasons for the progessive hypoxia and carbon dioxide accumulation in respiratory depression and emphysema. It explains the effectiveness of properly applied controlled or assisted breathing.

We have so far assumed the inspired gas mixtures to be evenly distributed within the lungs. This may be approximately correct in the healthy individual. In many conditions, like asthma, emphysema, pulmonary fibrosis, where the elasticity of the lung tissue deteriorates and local bronchiolar obstruction develops, we will find areas where the blood in the pulmonary capillaries is not properly oxygenated. Such uneven

distribution and ventilation leads to admixture of unoxygenated blood into the arterial blood stream, to reduction of the overall oxygen tension and arterial oxygen saturation. Even breathing 100% oxygen will fail to correct this type of hypoxia and restore efficient ventilation. The adequacy of pulmonary ventilation depends on numerous other factors—such as density and viscosity of the inspired gas mixtures, whether the airflow is smooth and laminary or turbulent—all of which tend to influence the intrabronchial resistance.

Up to now we have discussed pulmonary function only in its ventilatory aspects. Ventilation can be measured with comparative ease and give us a good estimate of patient's reserve-information adequate for most procedures in general surgery. However, adequate ventilation and air mixing do not assure per se adequate tissue oxygenation and carbon dioxide removal.

Diffusion

Diffusion is the next aspect of the pulmonary function that comes into play after a gas volume has been moved into the alveolar space. Diffusion is the process of gas exchange through semipermeable membranes. The exchange of oxygen and carbon dioxide through the alveolar epithelium has until recently been of more interest to the physiologist than to the clinician. We know that CO_2 is about 20 times as diffusible as oxygen and that, therefore, all clinical abnormalities of diffusion manifest themselves early as impaired oxygen uptake and not as CO_2 accumulation. We know furthermore that diffusion is impaired in such conditions as Boeck's sarcoid of the lung, berylliosis, asbestosis, scleroderma, certain gas poisoning and neoplasms. The measurements of this function involve arterial and alveolar gas analysis after breathing reduced oxygen percentages, etc. We really are only just beginning to gather information about diffusion, and this knowledge is at this stage not too useful in our field.

Pulmonary Capillary Blood Flow

Unless the pulmonary capillary blood flow is adequate as to volume, pressure and speed of flow, as well as evenly distributed, the overall pulmonary efficiency of oxygen uptake and carbon dioxide removal will be impaired. The effects will be the same whether this disturbance of the flow is due to inadequate pumping, as in right-sided heart failure, or to an intracardiac or intrapulmonary shunt, as in ventricular septal defects, cardiac congenital anomalies, pulmonary hemangiomas, atelectasis or pneumonias. There will be hypoxia, carbon dioxide accumulation and pH shift towards acidosis not correctable in all cases by oxygen administration. Certain of these conditions, like cardiac malformation or hemangiomas, can sometimes be surgically corrected, and it is in these conditions that a physiology laboratory will assist greatly in evaluating the cardiopulmonary function through arterial and alveolar gas analyses obtained by cardiac and pulmonary artery catheterization. A simple method might be developed from these studies some day that will help us obtain a continuous reading of the patient's arterial and alveolar oxygen, carbon dioxide, and blood pH during anesthesia. The emphasis is on simple, foolproof, and inexpensive equipment; the tests and equipment we have now do not satisfy these requirements.

Summary

We have superficially discussed the different aspects of pulmonary function, a function with which we interfere day in, day out, during the conduct of our anesthesia. I have tried to show you how delicately balanced the mechanics and the physiochemistry of breathing are, and how any disturbances of either ventilation, diffusion, or pulmonary capillary blood flow lead to the same combination of hypoxia, carbon dioxide accumulation, and respiratory acidosis. I have tried to point out how inadequate sometimes our means of evaluation are, how carbon dioxide might accumulate in a depressed patient breathing oxygen without assistance. I feel that our duties are not limited to the avoidance of cyanosis and surgical shock. I realize that many of the tests to evaluate pulmonary function are at this time not available to most of us. Constant attention and observation of our patients, however, can be practiced by all of us and will have to be continued, no matter what tests and apparatus may become available in the future. And remember, hypoxia kills quickly, but carbon dioxide and acidosis, however insidious, may be just as fatal.

The knowledge of pulmonary function provides a guide for the treatment of numerous nonsurgical diseases, like asthma, pulmonary fibrosis and emphysema. It gives us a picture of a patient's pulmonary reserve and thereby can help us in the conduct of safe anesthesia.

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Effect of Operative Positions On Vital Signs During Anesthesia

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Many times we have wondered why a patient's blood pressure has fallen, or his pulse pressure has narrowed, when he has been turned on his side, abdomen, or into some other operative position. Is it normal for this to occur, or is it due to the anesthesia, or to the position?

Some investigators, whose research on postoperative complications have included studies on vital capacity following abnormal positions, believe that malpositions may be the cause of many surgical fatalities.

With the view of accumulating additional data on the effect of operative positions on vital signs during anesthesia, I recently conducted a series of experiments in which I recorded the blood pressure, pulse, and respiration of 38 normal, healthy adults in the most common operative positions. All were unsedated and unanesthetized. The following is a report of my findings.

First, the blood pressure, pulse and respirations were checked with the patient in the supine position. The subjects were allowed to remain in the supine position for 20 minutes at which time their blood pressure, pulse and respiration was again checked. These readings—checked after the 20-minute period of relaxation—were considered to be normal values for this experiment.

Lateral Position

In this position, systolic pressures dropped from 0 to 22 mmHg** with an average fall of 11 mm. Diastolic readings dropped from 2 to 20 mm—with the exception of one person whose diastolic reading rose 12 mm. Pulses decreased 3 beats per minute. Respirations showed very little change.

Subjects were kept in the lateral position for 10 minutes. At the end of this period, systolic pressures had dropped an average of 17 mm, and diastolic, an average of 8 mm. Pulses dropped an average of 13 beats per minute. Respirations increased approximately 4 per minute.

Readings changed in direct proportion to length of time the position

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**Hereafter mm will refer to mmHg.

was maintained, and to the weight of the individual.

Lateral Position With Kidney Rest Elevated

While subjects were still in the lateral position, the kidney rest was elevated for a period of 15 minutes. Here the greatest incidence of fall in blood pressure occurred as soon as the ridge was raised, with very little subsequent change noted. Drops in systolic pressure ranged from 8 to 50 mm with an average fall of 25 mm. Diastolic pressures dropped only 10 mm. In all instances, pulse pressures narrowed. Pulse rates became slower by about 8 beats, and respirations increased 5 breaths per minute. All subjects stated it was difficult to breathe in this position; two complained of head pounding, and one complained of precordial pain.

Even after the kidney rest had been lowered for 5 minutes blood pressures had not returned to normal, the patients remaining somewhat hypotensive for 15-20 minutes. During this period, systolic pressures ranged from 6 to 22 mm lower than normal; diastolic pressures averaged 10 mm above normal. Pulse beats stayed within normal limits, as did respirations, except for one patient.

Prone Position

In this position, patients' readings were taken at the end of 5 and 10 minute intervals. At the end of 5 minutes, the following changes were noted: Systolic pressures dropped 5 mm and diastolic dropped 6 mm. Pulses dropped 7 beats per minute. Respirations remained unchanged, except in two instances. At the end of 10 minutes: systolic pressures dropped an average of 4 mm, and diastolic pressures dropped 6 mm. Pulses decreased 6 beats per minute. Respirations remained unchanged. This was the most comfortable of all positions.

Trendelenburg Position

Positions tested were from 5 to 20 degrees. After 5 minutes in the 10-degree position, the subjects' systolic pressures dropped an average of 8 mm. Diastolic pressure drops averaged 6 mm. Pulses decreased 7 beats per minute. Respirations changed only 2 breaths per minute.

Placed in a 20-degree position for an additional 10 minutes, the subjects' systolic pressures dropped an additional 4 mm. Diastolic pressures elevated 13 mm, with the end result of very narrow pulse pressures. In two instances pulse pressures narrowed from 50 mm to 16 mm. Here again, changes were in direct proportion to length of time the position was maintained, and to the weight of the individual. In any degree of Trendelenburg position, respirations changed little in rate, but a decrease in depth was noted. Subjects found it more difficult to breathe when placed in the severe, rather than in the moderate degree of Trendelenburg position.

Kraske's or Jackknife Position

In this position a pillow was placed under the chest to facilitate chest expansion. In half the cases tested, the blood pressures remained essentially unchanged when patients were placed in this position. In the other half, pressure rose immediately, the systolic pressures increasing an average of 15 mm, and diastolic pressures, 10 mm. Pulses became more rapid in all but two cases, the average increase being 7 beats per minute. Respirations increased only slightly.

Sims' Position

Systolic and diastolic pressures maintained their normal levels when this position was first assumed. Pulses increased by 8 beats. Respirations stayed the same.

Checked again after a 15-minute interval, readings showed systolic pressures to be unchanged. Diastolic pressures rose 7 mm, in all but one case. Pulses, in most instances, increased about 7 beats per minute. Changes in respirations were not remarkable. In this position, subjects showed the least amount of change in all vital signs.

Fowler's Position

When patients were first placed in this position, their systolic pressures tended to fluctuate. Pulses increased by 11 beats, and respirations increased by 11 breaths. Patients were kept in this position for 10 minutes, at the end of which time systolic readings had dropped an average of 26 mm, and diastolic an average of 5 mm. Pulse rates increased 5 beats. Respirations had a tendency to decrease slightly. The most severe drop in systolic pressure occurred in the obese patient.

Lithotomy Position

In this series it was noted that the patients' systolic pressures rose 5 mm while their diastolic pressures dropped 8 mm. Pulse beats and respirations changed very little. Rechecked after a time lapse of 15 minutes, readings remained unchanged, except for a drop in diastolic pressures which averaged only 4 mm. When patients' legs were lowered, systolic pressures increased an average of 10 mm. Diastolic pressures, pulse beats, and respirations stayed the same.

Case Histories

The following case reports from our hospital records are a few recent examples that illustrate clinical manifestations of the above findings.

Case 1.—A 40-year old female was scheduled for a ureteral lithotomy. Her history was essentially negative except for the ureteral stone. Hemoglobin was 9.5 gms and blood pressure, on physical examination, was 144/92. For premedication she was given nembutal gr. 3/4, demerol 100 mg, and atropine gr. 1/150. On arrival in the operating room her blood pressure was 145/100, pulse 84, and respirations 16. She was given a spinal anesthetic with 12 mg. 0.3% pontocaine with adrenaline. Prophylactically she was given 10 mg. vasoxyl and the level of anesthesia was to T-6. When the level had been fixed for 20 minutes, she was turned into the right lateral position. The systolic blood pressure immediately dropped 20mm; the other vital signs remained the same. Ten minutes later the kidney rest was elevated and the systolic pressure dropped 24 mm, a total of 44 mm. At this time the diastolic pressure dropped 20 mm, the pulse decreased 4 beats per minute and respirations increased 4 breaths per minute. The procedure lasted 1 hour and 15 minutes with the blood pressure staying down until an additional vasopressor was administered. As soon as the patient was turned to the supine position the blood pressure returned to the preoperative level and remained stationary.

Case 2.—A 28-year old female with cord compression at L-1 was placed in the prone position for a laminectomy. She had inactive tuberculosis, hemoglobin 12.5 gm., and blood pressure, on physical examina-

tion, was 98/72. For premedication she was given demerol 75 mg. and atropine gr. 1/150. She arrived in the operating room with blood pressure of 110/80, pulse 120, and respiration 18. For intubation she was given pentothal and quelicin and the anesthesia was maintained with ether and oxygen. The blood pressure remained at 108/80, pulse 100-112, and the respiration 16. After 2 hours the blood pressure dropped to 100/80, a fall of 8 mm. The pulse remained at 100, and the respiration at 16 until the operation was ended, 1 hour and 45 minutes later.

Case 3.—A 54-year old male patient with a history of essential hypertension, minimal myocardial damage evident by electrocardiogram, hemoglobin 12 gm. and blood pressure 180/110 on physical examination, was operated on for carcinoma of the rectum. An abdominal-perineal resection was performed. For premedication he received nembutal gr. 1½, demerol 100 mg. and atropine gr. 1/150. He arrived in the operating room with blood pressure of 140/90, pulse 84, and respiration 18. Intratracheal intubation was done following induction with pentothal and cyclopropane. Anesthesia was maintained with cyclopropane, oxygen and mecostrin. Preoperative levels of blood pressure, pulse, and respiration were sustained for the first hour in the supine position. He then was placed in 15-degrees Trendelenburg position and his systolic pressure dropped 40 mm in a 15-minute period. The diastolic pressure dropped only 20 mm. Pulse decreased 8 beats, and the respiration remained unchanged. The table was leveled and his systolic pressure rose 20 mm; diastolic, 10 mm. He was then given a vasopressor and his pressure returned to the preoperative level. For the perineal procedure he was placed in Kraske's position. The systolic pressure immediately dropped 20 mm; the diastolic rose 10 mm. Pulse increased 10 beats, and respiration 4 breaths, per minute. Throughout this period blood was adequately replaced according to weighed loss. When the patient was turned to the supine position at the end of the procedure, his blood pressure was 160/100, pulse 92, and respirations, 24.

Case 4.—A 43-year old, obese male was placed in the Kraske's or jackknife position for a hemorrhoidectomy. Preoperative examination showed 12 gm. hemoglobin, blood pressure 120/75, a history of being a chronic smoker and an alcoholic. For premedication he received nembutal gr. 1½, demerol, 100 mg. and atropine, gr. 1/150. Preanesthetic findings were, blood pressure 125/78, pulse 88, and respiration 20. A saddle block spinal was administered with 6 mg. .2 percent pontocaine. After being placed in Kraske's position the blood pressure was 130/100; pulse and respirations unchanged. The blood pressure level was maintained for the 30-minute procedure, and dropped to the preoperative level when he was again placed on his back.

We have modified our Kraske position because patients complained of severe abdominal pain when an acute position was used. Formerly, when we used a severe jacknife position, patients' blood pressures would rise higher with a more marked fall in systolic pressure when the table was leveled.

The majority of cases which require high Fowler's position in our hospital are neurosurgical. These procedures usually are performed with local infiltration of procaine. The patients generally have a slight ele-

vation of both systolic and diastolic pressures when they first are placed in this position. Pulse rates increase slightly, as do respirations. Systolic pressures drop sharply when patients are leveled. Diastolic pressures drop very little, as do respiration. Pulses increase a moderate amount.

Case 5.—A 63-year old man with history of tuberculosis, productive cough, hypertension, 200/120, grade 2 systolic murmur at the apex, benign prostatic hypertrophy, and 14 gm. hemoglobin, was placed in the lithotomy position for transuretheral resection of the prostate. For premedication he received nembutal gr. 3/4, demeral 75 mg., and atropine gr. 1/150. When he was brought to the operating room, the blood pressure was 200/140, pulse 108, and respiration 20. He was given a spinal anesthesia using 6 mg. 0.3 percent pontocaine, the level being established T-10. There were no changes in his vital signs. After 20 minutes, he was placed in the lithotomy position. The systolic blood pressure remained unchanged, but the diastolic pressure dropped 30 mm, and his pulse 20 beats per minute. These levels were maintained until near the end of the procedure at which time the patient was placed in a 10-degree Trendelenburg position. In this position his blood pressure rose, systolic 10 mm. and diastolic 40 mm; his pulse increased 12 beats, and respirations increased 8 breaths. The patient became very short of breath and cyanotic after 15 minutes and the table was leveled. Immediately his respiratory difficulty cleared, and blood pressure and pulse returned to preanesthetic levels within 5 minutes.

Overholt Position

I was unable to test the Overholt position on the unanesthetized patient, although it is being widely used in surgery. For several years, all lobectomys and pneumonectomys done by us at the tuberculosis hospital have been done in this position. During this period we have noted marked changes in the vital signs. Since the only records I have are on the anesthetized patients, I was unable accurately to tabulate the average change. In our experience, all such patients have a very marked narrowing of pulse pressure. The diastolic pressure usually elevates twice as much as the systolic, and the majority of patients maintain pulse pressures of 15 to 20 mm. The pulse becomes much weaker in volume, but the change in rate is not significant. Respirations decrease in depth and rate. If the blood loss is accurately replaced, these patients immediately maintain their preoperative vital signs when turned to the supine position at the close of the procedure.

How significant are these findings to the anesthetist? In studying these data there was evidence of circulatory and respiratory impairment, depending upon the acuteness of the position and the size of the individual. This indicates that we must take measures to compensate for these changes.

Of prime importance is the patient's respiration. This must be assisted whenever it is indicated. If a decreased respiratory rate and depth is encountered, and allowed to continue over a period of time, there will be decreased output of carbon dioxide from the lungs, thereby predisposing to respiratory acidosis. On the other hand, if the output of carbon dioxide is increased as a result of increased respiratory rate, respiratory alkalosis may occur.

One of the doctors has been making a series of tests to determine acidosis

and alkalosis on the surgical patients from whom blood samples are drawn before, during, and following surgery. It has been interesting to note that in all patients whose respirations have been controlled during surgery, the pH has been normal. Those whose respirations have been unassisted, have been either in acidosis or alkalosis.

When the blood pressure is below normal it indicates that the heart beat is weak, the blood vessels are relaxed, and the total quantity of blood in the vessels is reduced. The pulse pressure is an indication of how well the heart is overcoming the resistance offered it, how successfully it is driving the blood to the periphery, and the condition of the arteries. If a patient develops hypotension, or a narrowing of the pulse pressure, he should be given some means of support. Blood that has been lost should be replaced. If, however, hypotension results from a position which cannot be modified in some way, a vasopressor drug may be indicated.

Since the nurse anesthetist is with the patient throughout the surgical procedure, she should assume some responsibility for the positioning. The position should be as nearly natural as possible and extremely exaggerated positions should be avoided. This is particularly important in the patient whose blood vessels are sclerotic, or who is obese. The patient's chest should not be taped to hold him in position. The position should be made as comfortable as possible for the patient and soft pads should cover or replace metal ridges whenever possible.

We all are guilty of negligence if we bring the patient from the exaggerated to the supine position too suddenly after the surgical procedure. Sudden changes in position more often result in marked fluctuation in vital signs than do slow, gentle changes. There usually is no need for haste and patients will better tolerate the malpositions if we level them gradually over a few minutes' period. Many times, when the patient is in steep Trendelenburg position, leveling of the table can be started while the surgeon is closing the abdomen. In this way, the table will be level by the time the skin is closed, and the pressure, pulse, and respirations not be affected by the change.

Conclusions

(1) Always assist respirations when necessary for proper carbon dioxide elimination. (2) Support circulation by proper administration of fluids or vasopressors when each is indicated. (3) Keep the operative position as natural as possible and avoid extreme exaggerations, particularly for patients who have sclerotic vessels, or who are obese. (4) Change positions slowly and gently, at the beginning, as well as at the end, of the procedure.

Plastic Surgery and Some Related Anesthesia Problems

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The need for plastic surgery in various forms has increased from many standpoints other than the original concept of repair of the congenital cleft lip and palate. The demand for immediate repair of traumatic injuries, radical resections of malignancies (especially around the face), cosmetic surgical procedures, early coverage of extensive burns, and the transfer of tissue from one part of the body to another for adequate coverage, has increased many fold.

Yearly increases in the horsepower of the automobile, the greater mechanization of industry, the more widespread participation in sports, the "do-it-yourself" trends, and the ever-existent narcissistic tendencies of wanting to look better—whether from the facial standpoint, or to fit into a new Dior line—have helped create tremendous progress in the field.

In the adult, the use of 2% novocaine infiltration has proved ideal for rhinoplasty and other facial cases, provided that the patient has been properly prepared psychologically and given adequate preanesthetic sedation. The addition of 8 drops of adrenalin to the ounce of novocaine helps prolong the anesthesia and diminish bleeding. To this solution is added the 150 turbidity-reducing units of hyaluronidase for the diminution of edema and its accompanying distortion.

Preanesthetic sedation with local anesthesia usually works well with the combination of a morphine derivative type drug and a barbiturate to combat any possible novocaine sensitivity. The amount given, of course, varies with the size and mental state of the individual patient.

Occasionally a situation may arise where local anesthesia is necessary in order to secure the cooperation of the patient. This was the case with a young Indian woman who lost her entire scalp in a bean separating machine. It was necessary to provide skin coverage for the entire circumference of the head. Consequently, there were no healed surfaces for her to lie on during a general anesthesia. In view of this fact, the wet dressings were kept on the patient's head while the proper number of split grafts were removed from the

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thighs under spinal anesthesia. The patient was then allowed to sit up on the operating table with her back bolstered. In this position, a procaine block of the entire scalp was done by injecting a circumferential band around the entire head below the level of the raw surface. This worked extremely well, and it was possible to sew the skin grafts on without any difficulty.

Following the healing of the split skin grafts from the thigh, a full thickness skin graft with a better matching color was taken from the supra-clavicular area. For cosmetic reasons, this was used to cover a part of the forehead. A wig was then used for completion of the case.

Other advantages of local anesthesia include:

1. Cough reflex is present.
2. Less vascularity is present, resulting in less blood loss and greater ease of operating.
3. Vasoconstrictor drugs can be used with greater safety.

Some disadvantages are:

1. It generally is not too satisfactory for extremely apprehensive, or for psychotic patients.
2. Local infiltration into areas of disease or inflammation is contra-indicated.
3. Local anesthesia is difficult to produce in areas of inflammation of massive scar formation.

The decision to use general anesthesia for children and for adults requires a careful weighing of all physical factors. Its use depends on an evaluation of the individual need of the patient, and a consideration for his surgical requirements. The ideal general anesthetic system for any oral surgical procedure would include:

1. Nasal or oral endotracheal anesthesia.

In the case of jaw fractures, the nasal route is best as it often is necessary to check occlusion. In some mandibular fractures the patient is wired in occlusion at the completion of surgery. In these cases, the nasal endotracheal tube is left in place until the patient is fully awake and able to handle his own oral secretions. In severe mandibular fractures (especially with compounding into the mouth), I prefer to have the nasal endotracheal tube placed while the patient is awake so as to prevent hemorrhage and laryngo-spasm during induction. This can be accomplished with the combination of preanesthetic explanation of the procedure to the patient, a good preanesthetic sedation, and a good anesthetist. No topical anesthetic is applied to the larynx. The pentothal needle and syringe are held in place in the veins, the nasal endotracheal tube rapidly introduced, and the pentothal then immediately injected. The patient usually does not mind, and often does not remember the short period of discomfort.

2. Non-explosive agents in the presence of spark.
3. Availability of adequate suction.
4. Knowledge of the materials that are immediately available should artificial respiration become necessary.
5. Adequate throat pack, or inflatable cuff on tube, for prevention of foreign materials, or blood, entering trachea.
6. Suction and thorough inspection of pharynx at conclusion of operative procedure.
7. Careful observation of patient postoperatively until consciousness returns.

The use of rectal pentothal 10 to 15 minutes prior to anesthesia has been of great help in allaying patient

fear and in giving smoother inductions at the Milwaukee Children's Hospital. When the soft rubber catheter attached to the pentothal syringe is introduced, the children are told they are having their temperature taken rectally. Many of the children require reconstructive surgery. In these cases it has been found that rectal pentothal, given prior to their going into the operating room, eliminates the problems of ether induction, even in children who must be brought back to the operating room several times.

Severe burn cases often require a considerable number of anesthetics. Consequently, if adequate preparations can be made in advance, such as having dressings and materials ready, much anesthesia time can be saved. Daily dressings may be necessary to prepare an extensive area for skin grafting. I.V. pentothal is an excellent anesthetic for this situation. However, it is best to use this only during the time of removal and replacement of the lowermost portion of dressing when the granulating surface is exposed. Placement of the bulky portion of dressing can be done without discomfort while the patient is awake.

Following the application of the skin grafts, anesthesia is no longer necessary since the patient no longer suffers pain after the raw surface is covered.

Fear is the greatest factor to combat in a burn case, especially in children. Here, half the battle is won if the procedures are made as painless as possible and a good psychological approach is used.

Endotracheal anesthesia is now used routinely in all babies having harelip and cleft palate surgery. Needless to say, with this procedure the possibility of aspiration is eliminated, and the ease of surgery is increased.

I should like to describe a very interesting experience which created quite a problem, and to explain how this problem was handled. Several years ago I was called to see a man who tried to commit suicide by cutting his throat with what must have been an extremely sharp butcher knife. When I arrived at the Milwaukee County Emergency Hospital, the patient was lying on the operating table with a gaping wound across the entire neck. In the depth of the wound was the posterior pharynx, and in the lower surface of the wound, the glottis and true vocal cords were sitting right out in the open. Both jugular veins were exposed and bulging, and appeared to be ominously thin. Obviously the patient could not be put to sleep by the face mask method because of the open pharynx. Nor could pentothal be used first because of the danger of laryngospasm from the amount of blood in the wound. Consequently, tracheotomy was performed under local anesthesia. As the tracheotomy tube was being inserted, both jugular veins blew out with the first cough. Fortunately, a pentothal syringe was in place, and the patient was almost immediately anesthetized. From then on, it was simply a matter of tying off the jugular veins and going ahead with the repair of the wound in layers. That was really a very special kind of situation which one doesn't often come across.

Conclusion

With the continued cooperation of the anesthetist and plastic surgeon, we are looking forward to the continuance of recreating cosmetics and function to the best of our abilities in both the congenital and acquired deformities fields.

Prevention of Brachial Plexus Paralysis in Relation to Anesthesia

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Among the accidents which may occur during surgery against which anesthetists should be on guard is that of brachial plexus paralysis, for although its occurrence is rare, it is a calamity when it happens.

Most of us have been fortunate in never seeing a brachial plexus paralysis case. However, as a preventive measure it is wise to be aware of the circumstances under which it may occur. According to current medical literature, brachial plexus paralysis is most likely to occur where the following conditions are allowed to persist:

1. Abduction of the arm at a more than 90 degree angle.
2. Turning the patient's head in the opposite direction from an adducted arm.
3. Improper placing of the gallbladder rest.
4. Insufficient padding on shoulder braces when the patient is placed in the Trendelenburg position.
5. Placing the shoulder brace too close to the neck.
6. Abducting the patient's arms and straining his neck when placing him face down.

A surgeon of my acquaintance performed an operation for removal of cancer of the rectum in a patient. The patient later developed a paralysis of the left arm. Eighteen months after surgery the patient died without ever having regained the use of the arm. Most cases of brachial plexus palsy, however, are transient and recovery takes place in a short period of time. The discomfort and inconvenience it causes the patient varies. Needless to say, however, anesthetists should be on the alert to avoid causing the patient even minor discomfort through carelessness or oversight on their part.

If the patient is properly placed and protected before the anesthetic is started, there is little likelihood that an accident will occur. On the other hand, the widespread use of intravenous anesthetics, blood transfusions, and various intravenous fluids make it more than ever important for each member of the surgical team to be on guard against injuring the

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star of the performance—the patient.

Because injuries to the brachial plexus are either of a stretching or compression type, the following conditions should be observed in order that injury to the brachial plexus during the surgery may be avoided:

1. Never abduct an arm more than 90 degrees, and be on the alert to see that the elbow is not pushed up above shoulder level.
2. If shoulder braces are used, be certain that they are broad and well padded, and are placed, if possible, before the anesthetic is started.
3. Be sure that shoulder braces are not placed close to the neck.
4. Never allow a patient to be suspended by the wrists.
5. Never turn a patient's head in the opposite direction from an adducted arm.
6. If a gallbladder or kidney rest is used, be certain that there is no strain on the patient's shoulders.

In conclusion I would like to say that even though preventable injuries to the unconscious patient on the operating table are rare, the reports of such injuries when they do occur, usually are suppressed, perhaps for medicolegal reasons. Since this tends to prevent our profiting from the experiences of others, we sometimes have to learn of such incidents through our own bitter experience.

Treatment for brachial plexus paralysis is practically nil. Some physicians advise splinting the affected arm and instituting physiotherapy treatment as soon as possible. It should be borne in mind that the injury is to a nerve and that recovery depends on the extent of the injury.

Injury usually is in one arm only, although it has been known to occur bilaterally. Fortunately, the condition usually is transient. Prevention seems primarily to be a matter of constant alertness and the exercise of care by surgical team members in seeing that injury to the unconscious surgical patient is avoided.

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Endotracheal Anesthesia For Tonsil and Adenoid Surgery

We feel that the intubation technic for tonsil and adenoid surgery offers very definite advantages over the insufflation method of anesthesia and we employ this technic on patients of all ages.

During insufflation, the greatest difficulties are maintaining a patent airway, and preventing the aspiration of foreign material. Intubation provides and maintains a patent airway thus eliminating the mechanical obstruction with its attendant hypoxia and hypercapnea which too frequently accompanies the insufflation method.

Intubation almost totally eliminates the hazard of aspiration—an inevitable occurrence with the insufflation method. I know this statement will bring forth remonstrance from proponents of insufflation so in rebuttal I would like to ask if they have made it a practice—as I have—to do trachial toilets following tonsil and adenoid surgery. Using a polyethylene tube I have suctioned out from 3-10 cc of blood in many cases. This material, if inhaled into the lungs, could have caused serious obstruction.

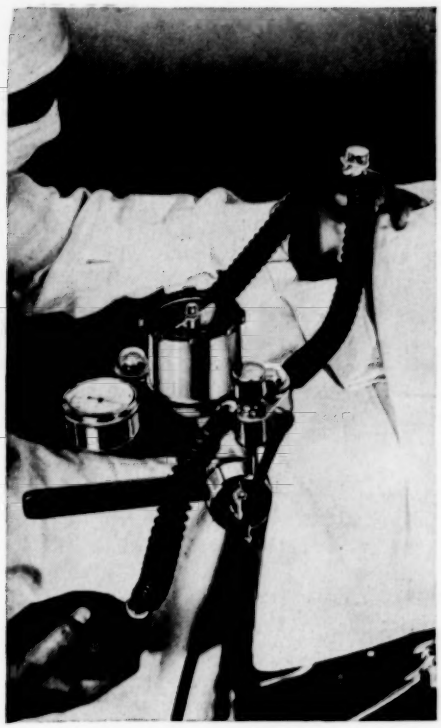
One time, for example, during a tonsillectomy performed by a surgeon who objected to intubation, the child rapidly developed dyspnea and became cyanotic. I intubated her at once and upon suctioning withdrew a large, well organized clot. With this removed, her respirations again became normal, her pulse and color good and we completed the operation without further incident. This surgeon has seen the light and is now a firm advocate of tubing.

During endotracheal anesthesia the anesthetist has a more complete control over the anesthetic than with other methods, the anesthesia being characterized by its smoothness.

The anesthesia is induced in the children with vinethene and ether. When the tube has been placed, anesthesia is maintained on cyclopropane, or on a very little amount of ether. We use the CO_2 absorption technic with the circle filter. Breathing tubes and bag are of children's capacity. With very small children we use the Ayres' technic. With this procedure we are able to have the patients react vigorously at the end of the case.

Our surgeons remove the adenoids with the tube in place. The anesthetist simply uses the curved end of the pillar retractor and raises the tube slightly while moving it over to the right or left side of the pharynx. This gives the surgeons ample room to insert their instruments. If they want the tube removed, the anesthesia is insufflated during the adenoidectomy.

With endotracheal technic the lungs may be quickly inflated with O_2. To do this during insufflation



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anesthesia, the mask must be placed over the operative field and as O_2 is forced into the lungs too often it is accompanied by blood and other secretions. In addition, with the mask over the face, the ability to suction, or to clamp bleeders, is temporarily lost.

When the patient is intubated, and the anesthetic is under control, the anesthetist is in a position to assist the surgeon when necessary. This method of anesthesia also gives the unskilled surgeon more time in which to complete his work unhampered by anxiety over airway, aspiration, pulmonary ventilation and prolonged deep anesthesia.

Finally—and worthy of consideration—personnel involved do not have to inhale quantities of ether from which they will reek for hours after the case is over.

Mrs. Marian Seguin
St. Vincent's Hospital
Portland, Oregon

Rectal Surital in Pediatric Anesthesia

In an effort to prevent the psychic trauma that too often accompanies the experience of anesthesia and surgery for children we at St. Vincent's Hospital, Portland, Oregon, are using rectal surital to provide basal hypnosis. The results have been very satisfactory.

Experience has taught us that giving 10 mg. of surital per pound of body weight results in a loss of consciousness which is not accompanied by any marked respiratory or circulatory depression, nor does the child have a prolonged recovery period.

The use of rectal surital in this dosage has not resulted in the loss of cough reflex, nor have we encountered laryngospasm which we could attribute directly to the barbiturate.

In preparing our rectal solution we use the rubber stoppered bottle which contains one gm. of surital. To this we add 20 cc. of H_2O making a 5% solution. Each cc. of this solution will contain 50 mg. of surital.

Children readily retain small amounts of this concentrated solution and a satisfactory degree of hypnosis is quickly achieved.

In calculating dosage we multiply the child's weight by 10. The resulting figure is divided by 50 (the number of mg. per cc. of solution). This resulting figure represents the number of cubic centimeters of stock solution that is to be given. For example: A 50 lb. child is to receive 10 mg. of surital per lb. of body weight. 10 mg. times 50 lbs. = 500 mg. of surital. 500 mg. divided by 50 mg. = 10 cc. of stock solution.

The paper on which the dose has been calculated is placed on the rectal tray which now contains the bottle of stock solution, a 20 cc. syringe, an 18-gage needle, a rubber catheter which has been cut down to 4″ in length, gauze sponges, lubricant, and 2″ adhesive. Tongue depressors and a pharyngeal tube are placed on the towel covering the tray.

At the bedside the desired dose is withdrawn from the bottle and the syringe is then attached to the lubricated catheter. After the child is turned on his side the tube is inserted and the solution injected under slight pressure by means of the syringe.

After the catheter is withdrawn the buttocks are taped together. In 10 minutes the child is usually so sleepy that he will not remember being

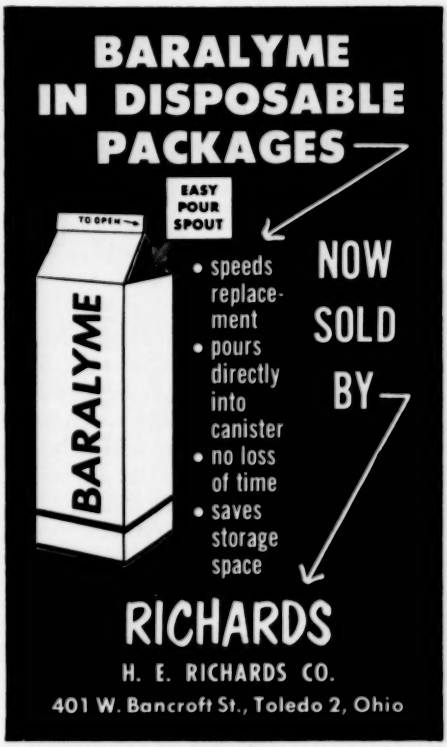
placed on the stretcher and conveyed to surgery.

By observing the following precautions we have avoided trouble:

1. To prevent respiratory obstruction, all children are placed on their sides until the general anesthetic is begun.
2. The basal anesthetic is never given until the surgeon is in the hospital.
3. Atropine is always used preoperatively.
4. Barbiturates are never used for asthmatic patients.
5. Oxygen, as it is for all unconscious patients, is always immediately available.

We do not consider the moderate doses of surital that are used to be hazardous.

MARIAN SEGUIN
St. Vincent's Hospital
Portland, Oregon



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Legislation

Emanuel Hayt, LLB., Counsel, A.A.N.A.

NURSE ANESTHETIST HELD NOT TO BE AGENT OF SURGEON DURING ANESTHESIA ADMINISTRATION—Plaintiffs brought this action against two defendants, a doctor and a hospital, to recover damages for injuries suffered by plaintiff wife during the administration of an anesthetic preparatory to a surgical operation in which her tonsils were to have been removed.

The trial court's instructions submitted to the jury the question of whether the nurse anesthetist (employed by the hospital) was the agent of the hospital or the agent of the doctor while administering the anesthetic to plaintiff.

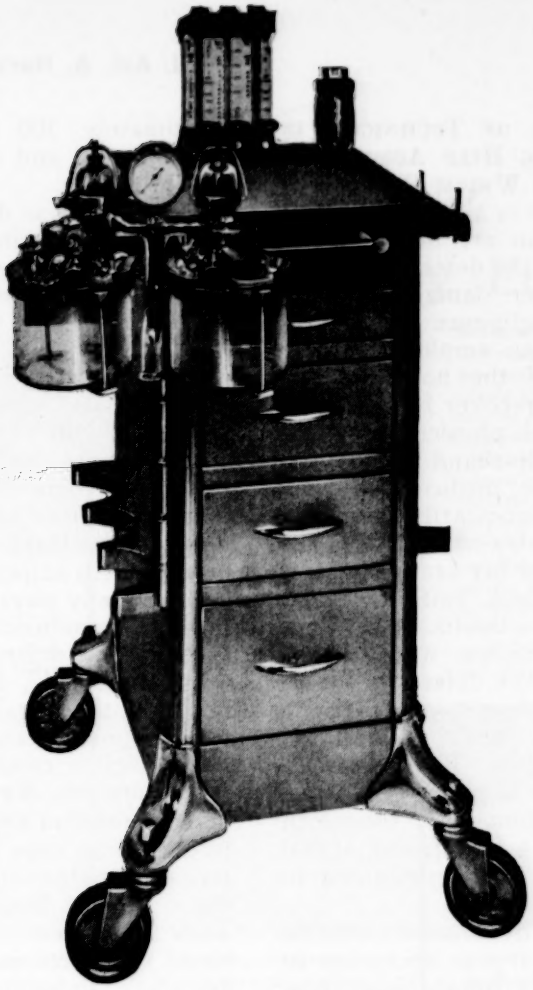
In the case at bar, the doctor's answer alleged that the nurse anesthetist was not his agent during the administration of the anesthetic. The testimony of the three surgeons as to the custom in Spokane hospitals constituted an evidentiary fact tending to prove that the doctor did rely on the nurse anesthetist and did not exercise any supervision or control over her actions as an anesthetist. These facts tended to prove the ultimate pleaded fact that she was not his agent during the administration of the anesthetic. Therefore, the trial court did not err in allowing the introduction of such testimony.

The trial court correctly submitted the issue of agency to the jury, since the evidence regarding it was in conflict. There was ample evidence from which the jury could, and apparently did, conclude that the nurse anesthetist was not the agent of the doctor, but was in fact the agent of the hospital, her employer. Normally, the hospital charged patients for the very services which the nurse anesthetist was rendering to plaintiff when this injury occurred. In the case at bar, the hospital made no charge to plaintiff for any services furnished her while she was in the hospital.

The doctor had testified that the nurse anesthetist knew considerably more about administering an anesthetic than he did, and that he left completely to her discretion the administration of the ether to the plaintiff. He also testified that all surgeons in Spokane customarily relied wholly on the nurse anesthetists or physician anesthetists supplied by the hospitals to administer anesthetics during surgical operations. Three other surgeons called by the doctor testified to the same effect.

A verdict for plaintiff, assessing her damages in the amount of $6,051.54 was returned against the hospital. The jury also returned a verdict for the doctor and against plaintiff. The judgment in favor of the plaintiff against the hospital is, therefore, affirmed.

(Kemalyan et al v. Henderson et al; Deaconess Hospital, Appellant, 277 P. 2d 372 (Wash.)



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Negligence of Technician in Blood Typing Held Administrative Act For Which Hospital is Liable.—This is an action by the plaintiffs, who are husband and wife, against the defendant hospital to recover damages arising out of the negligence of a laboratory technician employed in the laboratory of the hospital. The wife sues to recover for pain and suffering, both physical and mental, and the husband sues to recover for the medical expenses which he necessarily incurred on behalf of his wife as well as for the loss of her services.

The wife was suffering from rheumatoid arthritis. She consulted a physician, who advised her to enter the defendant's hospital for treatment. She became a patient at said hospital on March 17, 1947. The physician decided upon a course of treatment and in connection therewith directed that a transfusion of 500 cc. of blood be administered to her.

Before the transfusion could be administered it was necessary to determine Mrs. Berg's blood type, including its Rh factor. A blood sample was taken and the necessary test was performed by the laboratory technician employed in the hospital's laboratory. The technician mistakenly reported that Mrs. Berg's blood was type A-Rh positive, whereas, in fact, her blood was and is type A-Rh negative.

On March 19, 1947, 500 cc. of Rh positive blood was infused into Mrs. Berg. On March 26, 1947, while she was being infused with another 500 cc. of Rh positive blood, she developed an unfavorable reaction after approximately 100 cc. had been administered and the transfusion was stopped.

Mrs. Berg was discharged from defendant's hospital on April 12, 1947. Shortly thereafter she became pregnant. In May or early June, 1947, Mrs Berg consulted her family physician, who confirmed the fact that she was pregnant and directed her to a laboratory in Elizabeth, New Jersey, where she resided, for the purpose of determining the type and Rh factor of her blood. When this test disclosed that her blood type was Rh negative she informed her family physician of the Rh positive transfusions she had received at the defendant's hospital.

In June, 1947, Mrs. Berg also informed the physician whom she had originally consulted about her arthritic condition that the laboratory in New Jersey had determined that she was Rh negative. At that time Mrs. Berg was receiving outpatient treatment at the defendant hospital. Her New York physician then had her blood rechecked in the defendant hospital's laboratory and this time it was reported as Rh negative.

The infusion of Rh positive blood into an Rh negative female causes antibodies to be created and she becomes sensitized. In the event that such a female should become pregnant with an Rh positive fetus, such antibodies will in all likelihood cause the death of the fetus. During the course of Mrs. Berg's pregnancy it was established that the fetus was an Rh positive one, since her titer index (the quantity of antibodies in the blood) rose substantially. She was advised that this

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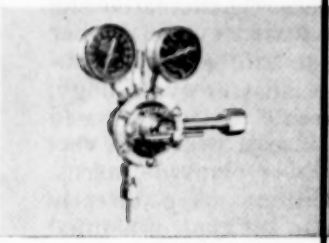

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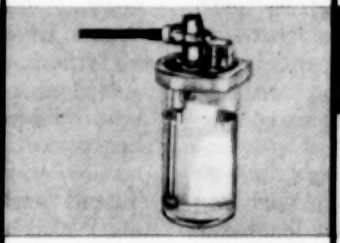

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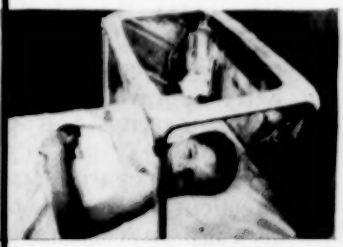

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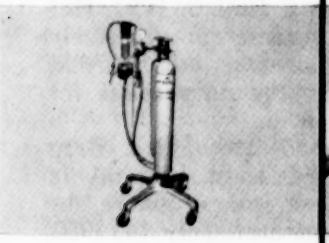

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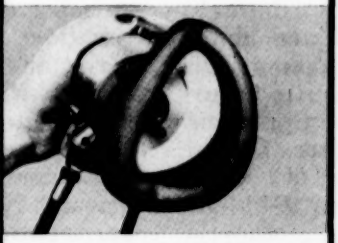

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increased titer would in all probability be fatal to the fetus. She was advised that the fetus died on December 2, 1947, but was further advised to carry through for the full period.

On December 30, 1947, Mrs. Berg was admitted to St. Elizabeth's Hospital in Elizabeth, New Jersey, where, after an entire day of labor, she delivered a still-born macerated female fetus on December 31, 1947. Undoubtedly the cause of the still-birth was the infusion of the Rh positive blood into Mrs. Berg. This wrongful infusion of incompatible blood into the mother created the blood disease of erythroblastosis fetalis, which was the competent and producing cause of the macerated fetus. Mrs. Berg had given birth to a son in 1944, who is Rh negative. The plaintiff husband is Rh heterozygous (his blood contains both negative and positive Rh factors.)

An Rh negative female has an excellent chance of carrying through uneventfully at least one Rh positive fetus, assuming, of course, that she has not previously been sensitized by a transfusion of Rh positive blood which to her would be incompatible, and she might even carry through successfully a second and third positive fetus pregnancy. Of course, an Rh negative fetus would never present any problem in this connection (see Gray's Attorneys' Text Book of Medicine, vol. 3, 3rd ed., ch. 304.14 to ch. 304.19)

The testimony is uncontradicted that prior to the transfusions received at defendant's hospital, Mrs. Berg had never received a transfusion.

In this case Mrs. Berg became highly sensitized by the transfusion of incompatible Rh positive blood. This sensitization undoubtedly caused the still-birth. The result was that she could not thereafter carry through successfully an Rh positive fetus.

Because of the circumstances, the attending physician at the delivery found it necessary to use manual pressure upon the abdomen for a much longer period than is usual or customary. This unusual and sustained manual pressure caused various internal difficulties which subsequently required medical treatment and ultimately an operation.

Mrs. Berg was discharged from St. Elizabeth's Hospital on January 4, 1948. Shortly thereafter she began to experience pain in her abdomen, side and back. She consulted a specialist in gynecology, who on March 1, 1948, diagnosed her condition as a cystocele, rectocele and a prolapsed uterus. These conditions were directly attributable to the type of manual pressure which was applied on her abdomen during the expulsion of the fetus and the placenta. Thereafter Mrs. Berg continued to receive medical treatment from this specialist until June 3, 1950. Her condition became progressively worse, and she was advised that surgery was the only relief for her condition.

On June 30, 1950, Mrs. Berg was admitted to a hospital in Newark, New Jersey, where she was a patient until July 14, 1950. During this period she underwent a vaginal hysterectomy to correct the prolapses of the bladder and the rectal wall. The condition of the uterus was such that it had to

be removed. At the same time her tubes were tied up, which was considered advisable. The effect of this surgical procedure was that she could not thereafter become pregnant.

On February 15, 1951, Mrs. Berg re-entered the hospital in Newark for the removal of a fistula of the vaginal wall, which condition was caused by the incomplete healing of the wound left by the previous surgical procedure.

From the time that Mrs. Berg discovered the effect that the transfusions would have upon her pregnancy she became nervous, irritable and apprehensive. It is clear that she suffered great mental anguish and a severe shock to her nervous system. This type of injury is of a permanent nature and was proximately caused by the transfusions of incompatible blood. Such type of injury is compensable under the circumstances.

The testimony adduced by the plaintiffs' doctors and expert witnesses supports plaintiffs' contention that the laboratory technician was negligent in performing the blood test to determine the Rh factor. The defendant offered no evidence to explain how the mistake occurred, but sought to elicit from plaintiffs' witnesses an admission that the Rh positive finding could have been caused by weak or impure serum purchased from outside laboratories. Plaintiffs' witnesses were in agreement and the court finds that weak or impure serum would result in an Rh negative finding but could never result in an Rh positive finding if the person being tested was in fact Rh negative.

The defendant contends that even if the laboratory technician was negligent it is not responsible because the performance of a test to determine the Rh factor of a blood sample taken from a patient is a medical act and not an administrative act.

Whether or not the testing of blood for the purposes under consideration herein by a laboratory technician employed in the hospital's own laboratory is a medical act or an administrative act is a question of law. The court is of the opinion that the test performed by the laboratory technician herein was an administrative act. She was not performing any medical act but was conducting a simple chemical test.

Taken in the light most favorable to the defendant the most that may be said of this test performed by the defendant's own employee is that it bore some relation to the field of medicine. It did not involve or require any decision on the part of the technician as to treatment or care of the patient. Ordinarily, a medical act would embrace the exercise of judgment as to the nature, character and symptoms of a disease, the determination of the proper remedy for the disease and the giving or prescribing of a remedy for the disease. None of these elements were present in the act performed by the defendant's laboratory technician.

A test to determine the Rh factor in a blood sample, when performed by a trained and competent technician, was a routine procedure in March, 1947. Accordingly, the court finds that the laboratory technician was guilty of negligence.

Although our courts have held that a hospital may not be held liable in damages for harm resulting from the negligent performance of medical acts usually performed by its doctors and nurses, nevertheless a hospital will be compelled to answer in damages for harm caused by the negligent performance of administrative acts by its servants and employees. This doctrine has even been extended to impose liability upon a hospital where doctors and nurses who are employees have been negligent in the performance of administrative acts.

A recent case which was decided on March 27, 1950, and which also involved the transfusion of incompatible blood as a result of which the patient died, is National Homeopathic Hospital v. Phillips (181 F., 2d, 293). In that case the court said: "The main question is whether such a relationship prevailed between the hospital and technician as to render the hospital liable upon the principle of respondeat superior. The trial court held a master and servant relationship did exist, and submitted the question of negligence to the jury, which returned a verdict for the plaintiff. We think the court was right. The undisputed evidence showed that the laboratory was an established part of the hospital. By arrangement with an outside physician it was operated under his overall direction. The technician was hired and paid by the hospital. In the instant case the hospital, in usual course, ordered a laboratory test. The technician, without the presence or supervision of the physician, made the test and submitted her report directly to the hospital. Relying thereon the hospital made the transfusion. In our opinion the facts clearly established the responsibility of the hospital for the acts of its technician. That responsibility is unaffected even though, agreeably to the requirements of 2 D.C. Code (1940) sections 101, 102 and 134 (b), the technical work in the laboratory was put under the direction of a physician."

In considering the damages to which the plaintiffs are entitled, the court leaves out of consideration any element of damages for the still-birth itself, since there can be no money recovery for the loss of an unborn child. Such damages are too remote and speculative to constitute an element proper for consideration either by a jury or by the court.

Defendant's motions to dismiss the complaint, made at the end of the plaintiffs' case and at the close of the entire case, upon which the court reserved decision, are denied. The defendant's motion for a directed verdict is also denied. Plaintiffs' motion for a directed verdict is granted.

It follows, therefore, that under the well established doctrine of respondeat superior, the defendant hospital may be compelled to respond in damages to the plaintiffs for all the harm caused by the negligence and carelessness of its employee, the laboratory technician.

The plaintiff Rose S. Berg is hereby awarded judgment against the defendant for $17,500. The plaintiff Sidney Berg is hereby awarded judgment against the

(Continued on page 215)

Book Reviews

Local Analgesia: Brachial Plexus. By R. R. Macintosh, M.A., D.M., F.R.C.S., F.F.A.R.C.S., D.A., M.D. (hon. causa), Buenos Aires and Aix-Marseilles, Nuffield Professor of Anaesthetics, University of Oxford, and William W. Mushin, M.A., B.S. (Lond.), M.R.C.S., F.F.A.R.C.S., D.A., Professor of Anaesthetics, Welsh National School of Medicine, University of Wales. Cloth. 3rd ed., 62 pages. 32 illustrations. Baltimore: The Williams & Wilkins Co., 1954. $3.00.

Except for a few minor additions, such as the effect of hyaluronidase on the duration of analgesia, this edition utilizes the material and illustrations of the earlier versions.

Well illustrated by drawings and photographs, the text is a concise, step-by-step guide for persons who perform this useful nerve blocking technic.

Nurse, Pastor, and Patient. By Granger Westberg, Chaplain, University of Chicago Clinics. Associate Professor of Pastoral Care, Federated Theological Faculty, The University of Chicago. 95 pages. Rock Island, Ill.: Augustana Press, 1955. $1.00.

The author's intent in this book is to give the nurse an opportunity of knowing how she may participate in giving spiritual comfort to patients of all creeds. The patient-nurse relationships, beyond the physical needs of the patient, are outlined in the first section of the text. The ways in which the nurse can help the pastor are the subject of the second section. Suggested prayers for use by patients and nurses, special verses from the Bible, and a list of books suitable for reading by patients, are some of the aids provided for nurses who wish "to enjoy the experience of a new dimension in caring for people who are ill."

Spinal Epidural Analgesia. By P. R. Bromage, M.B., B.S.(Lond.) F.F.A.R.C.S., D.A., Consultant Anaesthetist, Chichester Hospitals Group; Portsmouth Hospitals Group; Ministry of Pensions Hospital, Cosham; Visiting Anaesthetist, King Edward VII Sanatorium, Midhurst. Cloth. 123 pages, 41 illustrations. Baltimore; Williams and Wilkins Company, Publisher, 1954. $3.75.

Although recognizing that use of curare has altered the usefulness of regional anesthesia, the author initiated the study of spinal epidural anesthesia "as a reaction from the limitations of the relaxant techniques." The material in this small book is based upon 1,000 cases given by the author during the last five years.

Anesthesia in General Practice. By Stuart C. Cullen, M.D., Chairman, Division of Anesthesiology, Department of Surgery, State University of Iowa Hospitals; Professor of Surgery (Anesthesiology), State University of Iowa College of Medicine. Cloth. Ed. 4. 312 pages, 37 illustrations. Chicago, Ill.: The Year Book Publishers, Inc., 1954. $5.00.

To those who are familiar with this valuable book, the fourth edition will be a welcome addition to the anesthetist's library. A new chapter on ventilation and the extensive revision of the chapter on muscle relaxants places the latest thinking of the author into print. Those unacquainted with the book would do well to know this valuable occupant of the anesthetist's bookshelf.

Abstracts

Gillespie, C. H.: Considerations in anesthesia for the asthmatic patient. South. M. J. **58**:392-297 (March) 1955.

"So many drugs have been introduced for the treatment of the patient with asthma during the last few years that it behooves us to be cautious in the combined usage of some of these preparations. . . . The usual opiates (such as morphine, codeine, methadon hydrochloride, and dilaudid), used as depressants, have a prolonged constrictor action on the bronchi and are contraindicated. . . . The belladonna alkaloids added to morphine in proportions greater than 1:25 are considered by some authorities to override the bronchoconstrictor effect of morphine. Pharmacologically, the bronchi may be dilated, but the already tenacious mucous exudate is thickened more by the addition of the belladonna group of drugs with the formation of firmer bronchial plugs. This could lead to death by strangulation. Demerol Hydrochloride seems to be the best analgesic and premedicant in the patient with bronchial asthma because of its sympathomimetic action. . . . Small doses of atropine may be added to the Demerol when used as a preanesthetic drug. Nisentil Hydrochloride, a piperidine compound, may be used for minor surgery because of its dilating action on the bronchi. Scopolamine produces too much drying of the secretions and depression of the sensorium to be used routinely as a premedicant. Atropine has long been used as a premedicant in the patient with bronchial asthma. It should be used in small doses for larger amounts cause inspissation of mucus in the lungs. Atropine seems to prevent the access of acetylcholine to the cells of the organs innervated by the parasympathetic nervous system. It is active against both acetylcholine and histamine. Atropine should be used more as a prophylactic agent in asthma as it is not effective in relieving an attack once it is in progress. . . . .

"Previously, it was assumed that the parasympathomimetic drugs should be eliminated from the anesthetic procedure. Experience, however, has proved that such agents can be used successfully during the anesthetic process in the patient with bronchial asthma. Bentolila has used cyclopropane anesthesia for the treatment of status asthmaticus with apparent good results. . . . Although it is not the agent of choice for routine use, we have used cyclopropane anesthesia occasionally and successfully for asthmatic patients with diabetes mellitus or liver disease who refused to have spinal anesthesia. A primary contraindication to the use of cyclopropane for the asthmatic patient is the increased need for epinephrine and other vasopressor drugs. . . . The ultra short-acting barbiturates, in the form of Surital Sodium and Pentothal Sodium, are said to produce constriction of the bronchi but, nevertheless, they have the advantage of inducting anesthesia with such a

short excitement period that they have proved effective in patients with bronchial asthma. Barbiturates have been used empirically for many years in the treatment of hypersensitivity states and benefit is probably due to the fact that barbiturates react with thiamine in such a way as to inhibit the synthesis of acetylcholine. Other valuable anesthetic agents which produce relaxation of the bronchial musculature are chloroform, Trilene, Vinethene, and ether. Chloroform is seldom used in America but still has a fair reputation in Great Britain. Chloroform and ethyl chloride, however, have the same disadvantage as cyclopropane when used with the sympathetic amines. Trilene (trichlorethylene) is used chiefly as an analgesic. . . .

"Vinethine is excellent for short procedures, especially in children, and as an induction agent for longer ether anesthetics. Its bronchodilating effect and high potency give it a cardinal place among the suitable agents for the asthmatic child. Because ether is an excellent sympathomimetic agent, it has proved itself superior in the treatment of status asthmaticus and as an anesthetic agent for surgical procedures. . . . Procaine and cocaine have been attended by beneficial results in bronchial asthma and in other hypersensitivity diseases. It has been found that many local anesthetics have antiacetylcholine as well as antihistamine properties. Due to its direct bronchoconstrictor action, Pontocaine may produce a severe reaction in the asthmatic patient. Before intubation, if topical anesthesia is necessary, Pontocaine should be administered after the induction of a general anesthetic. The bronchial musculature is unaffected by nitrous oxide or ethylene unless hypoxia is present. . . .

"Occasionally stimulants must be used during the course of an anesthetic, Epinephrine has long proved to be powerful and constant in its sympathetic response. . . . Ephedrine may be used in the asthmatic patient for the purpose of combating low blood pressure or breaking an asthmatic attack. . . . Neosynephrine and Vasoxyl. . . .may also be used satisfactorily in the asthmatic patient. Because it has little or no direct action on the heart muscle, Vasoxyl is probably the only stimulant to be used if cyclopropane is the anesthetic agent. Norepinephrine (4 mg. per 1000 cc. of a 5 per cent dextrose solution) may be used very satisfactorily in the later and severe stages of shock. It lacks the degree of bronchial dilatation produced by epinephrine but when given at a sufficient drip controls the blood pressure at the desired level. In obstetrics, pituitrin is contraindicated because of its bronchoconstrictor action. Pitocin, however, can be used as it acts only on the pregnant uterus. . . . Excitement, in itself, has been known to be a factor in the production of an asthmatic attack. It is for this reason that a quick and easy induction is advantageous in the asthmatic patient. Where venipuncture is easily accomplished, it is best initiated by Pentothal Sodium or Surital Sodium induction. . . . In the child, either Vinethene induction or induction with rectal Avertin is best. Avertin produces bronchial dilatation and is pharmacologically sound for the asthmatic patients. It is contraindicated, however, in most other pulmonary diseases and in renal diseases. Rectal Pentothal may be used with care in the child with mild asthma but not in severe asthma. After induction, anesthesia is maintained with ether and oxygen. In the asthmatic patients

uncomplicated by heart disease, mild intermittent positive pressure is applied to adequately ventilate the patient. . . . If relaxation is needed over and above the ether, or if laryngospasm or bronchial spasm occur, it can be controlled by succinylcholine which first constricts and then relaxes the bronchus in a few seconds . . . . High spinal anesthesia should be eliminated, especially in the patient with severe asthma, because of the possibility of excessive secretions causing atelectasis. . . . The antihistaminics have little use during the anesthetic procedure. . . . The adrenal corticoids have opened new fields in the handling of the asthmatic patient, but the patient who has been receiving cortisone or ACTH therapy presents a very definite anesthetic risk. . . .

"Stoesser and Cook, cited by Curry, noted that large amounts of dextrose and water helped children with asthma and that the addition of saline negated the improvement. It is known that glucose stimulates the adrenals and thereby reduces the possibility of bronchial difficulties through increased circulating epinephrine. Rusk and his co-workers, cited by Curry, have reported a high serum potassium in asthma and improvement as the serum potassium falls. It would seem that fluid therapy should be limited to dextrose alone or dextrose in very small concentrations of saline. . . . It is said that complications occur three times more frequently in the asthmatic patient than in other types of patients. Here again, it is of necessity that all specialties concerned must be coordinated for handling the patient during this critical period . . . . In the early stage of asthma where secretions are the predominant factor, little more than the usual precautions are necessary. . . . During the second stage of chronic asthma, where bronchial swelling due to edema and corrugations of mucosa are present, the above measures should be supplemented with positive pressure breathing. . . . Oxygen-helium mixtures may be used if asthmatic breathing is evident. Should over-medication occur with the narcotic group of drugs, N-allylnormorphine (Nalline) may be effectively used without increasing the hazards of the asthmatic patient. It must be borne in mind that normorphine in itself may produce narcosis after antagonism is complete.

. . . Arnold has shown that pulmonary hypertension, which is sometimes a most troublesome factor, is easily controlled by expiratory positive pressure breathing with the O.E.M. meter mask. It is possible that a ganglionic blocking agent such as Arfonad (R2-222) may help to reduce the pulmonary hypertension more quickly if used along with positive pressure. This is still in the investigative stage. Along with the use of positive pressure, and a very important part of the treatment, is postural drainage. The lung must be kept as dry as possible. Little postoperative medication is necessary and then an agent which will produce only bronchial dilatation such as Demerol is recommended."

Classified Advertisements

NURSE ANESTHETIST: Additional Nurse Anesthetist wanted for new modern 217 bed general hospital. Full-time Anesthesiologist. No obstetrics. Liberal benefits and policies, salary open. Write details to: Director, Department of Anesthesia. Mercy Hospital, 144 State Street, Portland 3, Maine.

NURSE ANESTHETIST: To increase present staff of 9 nurse anesthetists. Starting salary $405 per month for members of A.A.N.A. with yearly increase plus laundry and private room with bath and telephone in new women's residence. Social Security and private pension plan. Excellent working conditions; surgical and delivery suite air conditioned. 40-hour week including call time. 1 month paid vacation. Apply: Marshall Kerry, M.D., Chief, Anesthesia, The Reading Hospital, Reading, Pennsylvania.

NURSE ANESTHETIST: Approved City-County Hospital. 400 beds. Few OB. cases. Three other nurse anesthetists with M.D. Anesthesiologist in charge. Starting salary $400. Inquire D. J. Walker, M.D., Anesthesiologist; Robert B. Green Hospital, San Antonio, Texas.

WANTED: Two nurse anesthetists, 300 bed hospital, attractive employment conditions; starting salary $400 per month. Night and weekend calls taken in rotation every fourth night and every fourth weekend. Six paid holidays annually. Minimal amount of obstetrical anesthesia. Full maintenance provided if desired. Approximately 5000 anesthesias administered annually for all types of surgical cases. Department is under supervision of an anesthesiologist. Apply to J. M. Schwab, M.D., Chief of Department of Anesthesiology, Geisinger Memorial Hospital and Foss Clinic, Danville, Penna.

SUPERVISOR OF NURSING SERVICE

WANTED: Supervisor of Nursing Service or Surgery Supervisor for this new fully equipped, amply staffed, 27 bed hospital 34 miles south of Memphis. Both hospital and beautiful nurses' home air conditioned throughout. Hours: Work 5 days, off 6th day. Two weeks vacation with pay. 12 days sick leave or a bonus if no sick leave taken. Social Security, retirement plan and Blue Cross. Applicant must be able to relieve anesthetist, otherwise could not be considered. Please furnish complete personal and professional data in first letter as well as least salary considered. Travel expense for interview will be furnished after mutual interest established. Write Murray E. Hill, Administrator, Tunica Hospital, Tunica, Miss.

NURSE ANESTHETIST WANTED: 500 bed teaching hospital on campus of state university. Starting salary $4512.00 with regular increases, cumulative sick leave, paid vacation. Apply to anesthesiologist in charge, University of Virginia Hospital, Charlottesville, Virginia.

THIRD ANESTHETIST wanted in approved general hospital of 184 beds in city of 25,000. Regulated hours and good working conditions. 4-weeks paid vacation. Paid sick leave. Salary according to qualifications. Minimum $400.00 per month plus full maintenance. Living accommodations in nicely furnished Nurses' Home. Apply Trinity Hospital, Minot, North Dak.

NURSE ANESTHETIST: For new 240 bed general hospital. Good salary, vacation, sick leave. Alternate emergency call with five others. 40-hour week. Attractive Southwestern city. J. E. Jenkins, Administrator, Bernalillo County-Indian Hospital, Albuquerque, New Mexico.

NURSE ANESTHETIST: For 117 bed fully approved hospital, located in the Shenandoah Valley. Desire third anesthetist to improve working conditions. Will also have anesthesiologist in the fall. Minimum salary $450 per month with allowance for experience. Reply Administrator, King's Daughters' Hospital, Staunton, Virginia.

ATTRACTIVE POSITION FOR NURSE ANESTHETIST: Apply to Leah Camp, M.D., Franklin Square Hospital, Baltimore 23, Maryland.

WANTED: Nurse Anesthetist for 800 bed teaching hospital. Staff of 4 Anesthesiologists, 2 Fellows, 4 Residents, and 9 Nurse Anesthetists. Beginning cash salary $4,512 annually with merit rating increases, one month paid vacation, 15 days sick leave annually, which can accumulate to 90 days; only emergency operations on Saturday. Please reply to Anesthesia Department, Medical College of Virginia, Richmond 19, Virginia.

REGISTERED NURSE ANESTHETIST: 135 bed general hospital in charming southern city of 18,000 short drive from Gulf of Mexico. Well-qualified surgical staff. Salary range $380-$416 a month commensurate with experience. 4 weeks vacation with pay, sick leave 2½ day weekend every 4th week. Apply: Administrator, John D. Archbold Memorial Hospital, Thomasville, Georgia.

REGISTERED NURSE ANESTHETISTS: 40 hr. week, permanent positions open for surgery and obstetric departments. Liberal vacation and sick leave policies. Social security, overtime pay, extra pay for night duty. No call duty. Automatic pay increases. Apply: Chief Nurse Anesthetist, Harper Hospital, Detroit 1, Michigan.

POSITION OPEN: Nurse anesthetist - excellent opportunity - 275 bed hospital. Apply: Robert M. Murphy, Administrator, Lima Memorial Hospital, Lima, Ohio.

NURSE ANESTHETIST - A.A.N.A. member. Two outstanding openings - one in Chicago, Illinois and one in Indiana. Good personnel policies; salary open; 40 hour week. Residential area. Contact E. D. Strzelecki Adm. Asst.-Little Company of Mary Hospital, Evergreen Park, Illinois.

NURSE ANESTHETIST: Approved hospital near Detroit. $475 per month. Overtime after forty hours per week. Living quarters available. Wyandotte General Hospital, Wyandotte, Mich.

NURSE ANESTHETIST: Needed immediately; new 250 bed hospital; expanding services; have anesthesiologist, four nurse anesthetists; need another to carry load. Salary $450 to $500 plus on-call fees; no maintenance. Contact Elmer W. Paul, Administrator, Methodist Hospital, Lubbock, Texas.

NURSE ANESTHETIST: 175 bed general hospital with only two nurse anesthetists at present. Air conditioned operating room; salary open; with or without maintenance; 5-day week; call two nights a week and every fourth weekend. No obstetrical work required. Apply: Mother Anne Gertrude, Administrator, Maryview Hospital, Portsmouth, Virginia.

NURSE ANESTHETIST: 150 bed general hospital, 150 bed TB hospital, in Guam. Two nurse anesthetists employed. Alternate nights and weekends on call.. Two-year contract at $3952 per annum plus 25% territorial post differential on Stateside recruitment; 40-hour week; overtime pay; annual increment about 5%; furnished new apartment available at $50 per month. Write: Director Medical Services, Government of Guam, Agana, Guam.

ANESTHETIST: 300 bed voluntary general hospital—not tax supported. New modern air-conditioned surgical suite just completed. Excellent working conditions. Live in or out. Five anesthetists on staff. Salary open. Apply Decatur and Macon County Hospital, Decatur, Illinois.

NURSE ANESTHETIST: Oral surgeon in the vicinity of Boston, Mass. would like nurse anesthetist beginning August 15, 1955. Leonard S. Fox, D.D.S., 69 B. Court St., Westfield, Mass.

POSITION WANTED: A.A.N.A. Male, married, two school children. Chief past 7 years. Desires position suburban N.E. commensurate with above responsibility. Will consider additional administrative duties. Available 30-day notice. Write Box E-25, Journal American Association of Nurse Anesthetists, 116 S. Michigan, Chicago 3, Ill.

ANESTHETIST: RNA — 340 bed accredited general hospital with immediate program 240 bed addition. City 250,000 near beaches. Salary $400 month. Room available. Medical Anesthesiologist in charge of department. Write, Director Anesthesia, Tampa Municipal Hospital, Tampa, Fla.

NURSE ANESTHETIST: For approved general hospital. Must be able to administer latest types anesthetic agents. Salary $350.00 to $375.00 per month plus full maintenance. Annual vacation and sick leave. Retirement benefits if desired. Apply: Administrator, Robinson Memorial Hospital, Ravenna, Ohio.

NURSE ANESTHETIST: New 80 bed general hospital, to open Sept. 1, new equipment, salary $500 per month, paid vacation, holidays; college town of 9,000. Apply Gratiot Community Hospital, Alma, Mich.

NURSE ANESTHETISTS: Good Salary, good working conditions. Apply Chief, Anesthesia Dept., The Mercer Hospital, Trenton, N. J.

MOVING TO PHILADELPHIA? Suburban general hospital, 130 beds, needs a nurse anesthetist in addition to present staff. Modern air conditioned surgery. Write Administrator, Chestnut Hill Hospital, 8835 Germantown Avenue, Philadelphia 18, Pa.

NURSE ANESTHETIST WANTED: For obstetrical anesthesia in department averaging 250-300 deliveries monthly. Salary $400 month plus complete maintenance, including room, meals, phone and laundry. Vacation and sick time granted. Apply: Peoples Hospital, Akron, Ohio.

NURSE ANESTHETIST: 600 bed approved general hospital; excellent salary, one month vacation after a year's service. Apply, Personnel Director, Good Samaritan Hospital, Cincinnati 20, Ohio.

NURSE ANESTHETIST: Registered Male or Female. 185 bed approved general hospital. Salary open plus meals. 40-hour week, 3 weeks vacation. Merit increases. Personnel Dept., Blessing Hospital, Quincy, Ill.

A position for a Nurse Anesthetist is available at the Cleveland Veterans Administration Hospital, 7300 York Road, Cleveland 30, Ohio. Applicant must have had training in an approved school and be a member of the American Association of Nurse Anesthetists. Department staff includes one physician and five nurse anesthetists.

Position available immediately. Permanent or summer relief - 200 bed hospital. Paid vacations, sick leave and Social Security. Medical Anesthesiologist in charge. Apply: Sister Mary Concetta, St. Joseph Mercy Hospital, Pontiac, Mich.

NURSE ANESTHETIST: Fully approved 83 bed hospital in town of 8000. Delightful year around climate. Able to handle all types of anesthetic agents. Two years experience required. Salary commensurate with ability. Full maintenance, liberal benefits. Paid vacation, holidays and sick leave. 4-room apartment available, furnished or unfurnished, if preferred to nurse's home. No obstetrics. Telephone collect, daytime, Newton 630, evening, 1850, W. C. Shoemaker, Administrator, Catawba Hospital, Inc., Newton, North Carolina.

ATTENTION
University Hospitals of Cleveland School of Anesthesia Alumnae

There will be no meeting of the alumnae during the AANA annual meeting in 1955. A meeting will be held in 1956.

NURSE ANESTHETIST: 120 bed hospital. 40-hour week with chance to take cases on time off. Pleasant working conditions. Salary open. Contact Director of Nursing Service, St. Joseph's Hospital, Nashua, New Hampshire.

NURSE ANESTHETISTS: For 130 bed general hospital; four nurses, two M.D.s. Active general surgery and OB. Rotate call and off days. Apply, Administrator, Salem General Hospital, Salem, Oregon.

NURSE ANESTHETIST: JCAH approved 65 bed general hospital. Light surgery schedule. No obstetrics. Maintenance. Good salary. The Marion Sims Memorial Hospital, Lancaster, South Carolina.

NURSE ANESTHETIST: 86 bed general hospital, A.C.S. approved; located in city of 18,000; 2 hours drive from Memphis, Tenn.; excellent nurses' home; $425.00, plus full maintenance; will raise to $450.00 no later than six months after satisfactory employment; rotate working shift with another anesthetist. Apply to: Admin-

WANTED: To complete staff of three istrator, Helena Hospital, Helena, Ark.

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serving 100-bed hospital. Nurse anesthetist. Excellent working conditions, vacation, sick leave, furnished apartment, good salary, no OB calls. Additional information on request. Pulaski Hospital, Pulaski, Virginia.

ANESTHETISTS: Charleroi-Monessen Hospital (general, 238 beds and bassinets), North Charleroi, Pa. (HU 3-5561). Salary open plus maintenance, sick leave, holidays, month paid vacation annually, etc. Call or write administrator.

NURSE ANESTHETIST: A.A.N.A. qualified. Excellent personnel policies. Remuneration in keeping with experience and training. Quarters available with complete maintenance. Consulting anesthesiologist in charge. Apply: Director of Anesthesia, Norfolk General Hospital, Norfolk, Virginia.

WANTED: Surgical Anesthetist for 150 bed general hospital central Nebraska. Excellent working conditions and personnel policies. $450 per month and full maintenance. Apply: Box M-28, Journal American Association of Nurse Anesthetists, 116 S. Michigan Ave., Chicago 3, Ill.

NURSE ANESTHETIST: For 230 bed, well equipped modern general hospital in Western Pennsylvania. Town of 50,000. Five anesthetists employed. Rotating call every fourth night and every fifth weekend. Day off following call. Salary $425. 28 days vacation. Sick leave and Social Security. No maintenance. Apply: Box E-27, Journal American Association of Nurse Anesthetists, 116 S. Michigan Ave., Chicago 3, Ill.

ANESTHETIST: Fifth anesthetist wanted in 186-bed accredited hospital. Regulated hours, vacation, sick leave. Salary according to qualifications—minimum $400 per month. Apply: Superintendent, Lutheran Deaconess Hospital, 1138 N. Leavitt, Chicago, Ill.

WANTED: Nurse anesthetist. 250 bed hospital. Salary with or without maintenance. St. Joseph Hospital, 2100 Burling St., Chicago, Ill., telephone Mohawk 4-1700.

NURSE ANESTHETISTS: Two nurse anesthetists needed for a 200 bed general hospital. Salary $500 per month. Apply C. K. Shiro, Administrator, Montana Deaconess Hospital, Great Falls, Montana.

NURSE ANESTHETIST: Position now available at the Veterans Administration West Side Hospital, 820 S. Damen Ave., Chicago 12, Ill. For further information contact Dr. Melvin Gibbel, Chief of Surgery at the above address.

WANTED: Two nurse anesthetists for 206 bed, non-profit, general hospital. One for obstetrical coverage, no surgery. One for surgical coverage, no OB. Salary open - with or without maintenance. Apply: Personnel Director, Petersburg General Hospital, Petersburg, Virginia.

ANESTHETISTS WANTED: Busy suburban hospital near Chicago. New nurses' residence. (Apartments available for married anesthetists). Starting salary $375 per month plus full maintenance. Attractive work schedule. MacNeal Memorial Hospital, Berwyn, Ill.

NURSE ANESTHETISTS: New 225 bed general hospital. Medium sized town 60 miles east of Pittsburgh. Excellent working conditions and personnel policies. Very good salary. Write: Robert L. Seifert, Personnel Director, Mercy Hospital, Johnstown, Penna.

NURSE ANESTHETIST: 106 bed general hospital in the process of expansion to 165 beds. Medium size university town, 70 miles from Atlanta. Salary depending on experience, plus full maintenance. Anesthesiologist on medical staff. One Nurse anesthetist presently employed. Apply: Administrator, Athens General Hospital, Athens, Georgia.

TWO NURSE ANESTHETISTS WANTED: 44-hour week. Surgery new and air-conditioned. No day work when taking calls. Attractive living quarters. Full maintenance at $24 per month. Salary and benefits according to State Civil Service. Apply to O. P. Daly, M.D., Superintendent, Lafayette Charity Hospital, Lafayette, Louisiana.

NURSE ANESTHETIST: Five days 40 hours, two weeks paid vacation, six paid holidays, on call every third week, $550 per month. Apply: Klamath Valley Hospital, Klamath Falls, Oregon.

ANESTHETIST: 400 bed general hospital; located in a delightful lake surrounded university city. Pleasant working conditions in modern surgical and obstetrical departments. Liberal vacation, sick leave, social security, group insurance, and pension plan. Salary $450. Apply: W. D. Barclay, Assistant Administrator, Madison General Hospital, Madison 5, Wisconsin.

NURSE ANESTHETISTS: Two needed at a small approved general hospital in Pittsburgh. Supervised by a qualified anesthesiologist. Salary open. 35 and under preferred. Apply: St. Margaret Memorial Hospital, 46th Street, Pittsburgh 1, Pennsylvania.

NURSE ANESTHETIST: Approved 131 bed general hospital, Bristol, Tenn. 44-hour week, alternate call with two other anesthetists. New, modern hospital, pleasant working conditions. Salary $375 per month with $25 increase at end of six months. Vacation and two weeks sick leave annually. Laundry, meals, and room in new nurses home. Write: Marigold Jones, Department of Anesthesia, Bristol Memorial Hospital, Bristol, Tennessee.

LEGISLATION

(Continued from page 206)

defendant in the sum of $2,500.

The defendant to have thirty days' stay of execution and sixty days to make a case.

The foregoing constitutes the decision of the court pursuant to section 440 of the Civil Practice Act.

Settle judgment.

(Berg v. N.Y. Soc'y for the Relief of the Ruptured and Crippled, Supreme Court, N.Y. County, Trial Term Part XX, Osterman, J., N.Y.L.J., 12-31-54, p.7)

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